ANALYSIS OF METABOLIC PARAMETERS PREDICTING OUTCOME IN POLYTRAUMA PATIENTS

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CERTIFICATE

This is to certify that this dissertation titled **"ANALYSIS OF METABOLIC PARAMETERS PREDICTING OUTCOME IN POLYTRAUMA PATIENTS"** is a bonafide record of work done by **DR.S.K.SARAVANAN,** during the period of his Post graduate study from JULY 2014 to April 2016 under the guidance of me in the Institute of Orthopaedics and Traumatology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-600003, in partial fulfillment of the requirement for **M.S. (ORTHOPAEDIC SURGERY)** degree Examination of The Tamilnadu Dr. M.G.R. Medical University to be held in April 2016.

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DECLARATION

I declare that the dissertation entitled **"ANALYSIS OF METABOLIC PARAMETERS PREDICTING OUTCOME IN POLYTRAUMA PATIENTS"** submitted by me for the degree of M.S is the record work carried out by me during the period of **July 2014 to September 2015** under the guidance of **PROF.N. DEEN MUHAMMAD ISMAIL, M.S.Ortho., D.Ortho.,** Institute of Orthopaedics and Traumatology, Madras Medical College, Chennai. This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the University regulations for the award of degree of M.S.ORTHOPAEDICS (BRANCH-II) examination to be held in April 2016.

This work has not formed the basis for the award of any other degree or diploma to me previously from any other university.

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INTRODUCTION

Trauma is a major worldwide cause of death and disability that mainly affects young adults and the elderly population(1). The definition of multiple trauma varies among surgeons from different specialties and between different centers and countries. Polytrauma patients are the subgroup of injured patients who have sustained injuries to more than one body region and organ with at least one of the injuries being life-threatening(2). For uniformity, polytrauma is defined as injuries with injury severity score more than 16(3). Trentz emphasized the pathophysiologic systemic impact of multiple trauma when he defined polytrauma as “a syndrome of multiple injuries exceeding a defined severity (ISS ≥17) with sequential systemic reactions (systemic inflammatory response syndrome [SIRS] for at least 1 day) that may lead to dysfunction or failure of remote organs and vital systems, which have not themselves been directly injured.”(4)

EPIDEMIOLOGY

According to WHO report, the global injury mortality rate is estimated to be 98/100,000 population. Five of the top ten causes of death globally are due to injuries(5). About 12.8% patients sustain severe injuries (ISS 16 to 24) and 9.6% patients suffer very severe injuries (ISS >24). Motor vehicle accidents account for 37% of all cases, followed by falls at 30%. Major peaks occur in 16 to 24 years because of motor vehicle accidents. Males are more prone to trauma. The predominant cause of death after trauma continues to be central nervous system (CNS) injury (21.6–71.5%), followed by exsanguination (12.5–26.6%), while sepsis (3.1–17%) and multi-organ failure (MOF) (1.6–9%) continue to be predominant causes of late death.(6)

POLYTRAUMA OUTCOME

An organized trauma care is necessary to reduce the mortality due to polytrauma, which consists of a good pre-hospital and in-hospital care and rehabilitation. Research and constant reevaluation are necessary for continuous assessment of the system and improvement of its outcome and efficiency.

The following table emphasizes the significance of various variables in deciding the final outcome in polytrauma patients.

TABLE 1 Comparison of Continuous Variables between Survivors and Those Who Died, by Age Group

	Elderly Adults			Adult		
	Survived, Median	**Died, Median**	**P**	**Survived, Median**	**Died, Median**	**P**
Age (yr)	74	76*	.013	33	35*	.045
Injury Severity Score	20	26*	.001	25	30*	.001
Glasgow Coma Scale score	14	9*	.001	14	3*	.001
Emergency fluids (mL)	2000	2000	NS	2000	2500	.001
Pulse (/min)	80	88*	NS	86	80*	.001
Blood pressure (mm Hg)	140	130*	.024	130	110*	.001
Respirations (/min)	18	18	NS	18	14	.001

* Statistically significant difference between elderly and adult patients who died (Mann-Whitney *U* test, $P<0.001$).

At present, the severity of the injury and need for intensive monitoring and care are decided based on the basic vital parameters and trauma scoring systems. Systolic BP less than 90 mmHg is

considered as shock and resuscitated intensely. According to a recent study, shock index is considered superior to systolic BP in diagnosing haemorrhagic shock(7). Shock index is defined as the ratio between heart rate and systolic BP. Shock index of > 0.9 is considered significant and intensive monitoring and care is given to combat occult hypoperfusion.

In an uninjured, healthy, nonseptic state, oxygen consumption is a regulated process because oxygen is used in the generation of energy from a variety of metabolic fuels(8). In case of occult haemorrhagic or septic shock, the oxygen delivery to the tissue is drastically reduced creating a crisis of ischemic metabolic insufficiency. When oxygen saturation reduces below the threshold limit, the tissues go in for a state of oxygen debt or oxygen deficit which is defined as the integral difference between the pre-trauma/pre-haemorrhage oxygen saturation and saturation during the hypovolemic, haemorrhagic phase. The presence of oxygen debt is further emphasized by the accompanying metabolic acidemia due to increased anaerobic activity. Metabolic acids in blood are indices that reflect the degree of tissue hypoxia associated with hypovolemic ischemia. In this review, the strict definition of base deficit (BD) – namely, a negative base excess – is used with a

decrease in base excess with increasing metabolic decompensation implying progressively negative values (e.g. –6 mmol/l to –10 mmol/l). However, because BD implies a negative base excess, only positive values of BD (without the minus sign) are used(9).

The base deficit is proved to be one of the best predictors of occult hypoperfusion(10). The admission BD was one of the five best predictors for outcome (BD, GCS, age, prothrombin time, and ISS)(8). Each of these five variables contributes significantly to the prediction of severity and mortality.

Of all the above said vital and blood parameters, we hypothesize that elevated serum lactate gives clear indication that the amount of oxygen in the blood is insufficient and causes debt in the tissues, thus having risk of multi organ dysfunction syndrome. In many of the studies, lactate was shown to predict outcome following postoperative complications, intracranial pressure, infection, sepsis, adult respiratory distress syndrome (ARDS), MOF, injury and haemorrhage severity and survival(11).

TRAUMA SCORING SYSTEMS AND THEIR VALUE

The need for comparative analysis of injury, management & outcome related parameters has stimulated the development of many

trauma scoring systems. These scoring systems represent a means of quantifying the injuries along with comorbidities, age and mode of injury. They are based on converting many independent factors into one-dimensional numeric value that ideally represents the criticality of the illness. An ideal scoring system should take into account the severity of anatomic trauma, level of physiologic response & inherent patient reserves.

Abbreviated injury score (AIS)(12) is an anatomically based, consensus derived, global severity scoring system denoting each body region with a seven-digit number code, the last digit being the injury severity. ISS(13) (Injury Severity Score) is the sum of the squared AIS scores for the three most severely injured ISS body regions. It can take value from 1 to 75. ISS represents the gold standard of anatomic trauma scoring systems as it have a linear correlation with mortality, morbidity, hospital stay and other measures of injury severity. The weaknesses of ISS are that it underestimates the overall anatomic injury in case of multiple injuries in one body region, and any error in AIS scoring increases the ISS error. Thus comes the NISS (New ISS) (14), wherein the sum of the squares of the three highest AIS severity scores is taken into account regardless of the ISS body regions.

Revised Trauma Score (RTS)(15) is a physiologically based scoring system taking into account GCS, RR & SBP, a coded value of 0 to 4 assigned to each, score ranging from 0 to 12 with lower scores representing a more critical status.

Table-2 Unweighted Revised Trauma Score as Used in Field Triage

GCS Score	RR (/min)	SBP (mm Hg)	Coded value
13 to 15	10 to 29	>89	4
9 to 12	>29	76 to 89	3
6 to 8	6 to 9	50 to 75	2
4 to 5	1 to 5	1 to 49	1
3	0	0	0

GCS, Glasgow Coma Scale; RR, respiratory rate; SBP, systolic blood pressure.

APACHE is the most modern scoring system taking age, any chronic health comorbidities, etc., into account(16).

The individual deficiencies of anatomic scales and physiology based scores led to development of combined scores, like TRISS(17) and ASCOT(18), which incorporates ISS and RTS, as well as patient's age.

PATHOPHYSIOLOGY AND IMMUNE RESPONSE

After injury, first there is a hypodynamic ebb phase (shock) where the body initially attempts to limit the blood loss and to maintain perfusion to the vital organs. This is followed by a hyperdynamic flow phase lasting for up to 2 weeks, which is characterized by increased blood flow to remove waste products and to allow nutrients to reach the site of injury for repair. The last phase is a recuperation phase that may last for several months in an attempt to allow the human body to return to its preinjury level(19).

The first physiologic response is the release of adrenocorticosteroids and catecholamines, named by Hans Selye as 'the general adaptation syndrome'(20). This is the forerunner of SIRS, responsible for the increase in heart rate, RR, fever and leukocytosis. The activation of immune system following a traumatic insult is necessary for haemostasis, protection against invading microorganisms and the initiation of tissue repair and tissue healing. Multiple alterations in inflammatory and immunologic functions have been demonstrated in clinical and experimental situations following trauma and haemorrhage, suggesting that a cascade of abnormalities that ultimately leads to adult respiratory distress syndrome (ARDS) and multiple organ

dysfunction syndrome (MODS) is initiated in the immediate post injury period(21)(22). Generalized hypoxemia following blood loss and tissue damage leads to damage of endothelial membranes, thereby activating circulatory immune system and subsequently activation of coagulatory system, complement system, prostaglandin system, and drop in platelet count.

Table – 3 Defined Parameters of SIRS	
Body temperature	> 38 °C or <36 ° C
Heart rate	> 90 beats/min
Respiratory rate	> 20/min or PaCO2 <32 mm Hg
White blood cell count	> 12,000 or <4000/mm3 or >10% band forms

The release of mediators of both a pro-inflammatory and an anti-inflammatory nature is dependent primarily on the ‘first hit phenomenon’ related to initial trauma and secondarily on the therapeutic and diagnostic interventions and postoperative complications, which are the second and third hits(23). Trauma induces local cells to release PMN granulocytes, monocytes & leucocytes which trigger a multifocal molecular and pathophysiologic process. The mechanism of complement activation, leukostasis and macrophage activation has been

associated with the concept of “low flow syndrome”(24) and more recently with endothelial and PMN leukocyte activation(25). The cells interact and adhere to the endothelium via adhesion molecules like L-Selectin & integrin B2. Toxic enzymes are released causing ARDS or MODS. Serum markers of immune reactivity are grouped into markers of acute phase reactants, mediator activity & cellular activity(26). Interleukin-6 is the most useful and widely used because of consistent expression and plasma half-life. A cutoff value of 200 pg/dl of IL-6 was shown to be significantly diagnostic of a “SIRS state”. Alarmins(27) are endogenous molecules triggering innate immune response mediators like heat shock proteins, defensins, cathelicidin and eosinophil derived neurotoxin. These are endogenous mediators of innate immunity, chemoattractants and activators of antigen presenting cells. High mobility group box 1 (HMGB1) is a nuclear protein that influences nuclear transactions and plays a role in signaling after tissue damage. Pathogen associated molecular patterns representing inflammatory molecules of a microbial nature & damage associated molecular patterns are recognized by our immune system by the expression of multiligand receptors such as Toll-like receptors. Overall, these molecules

represent a newly documented superfamily capable of activating innate immune responses after trauma.

Table-4: Serum Inflammatory Markers

Group	Serum Inflammatory Markers
Acute phase reactants	LBP, CRP, procalcitonin
Mediator activity	TNF, IL-1, IL-6, IL-10, IL-18
Cellular activity	TNF-RI, TNF-RII, IL-1RI, IL-1RII, sIL-6R, mIL-6R, ICAM-1 E-selectin, CD11b Elastase, HLA-DR class II antigens, DNA

LBP, lipopolysaccharide-binding protein; CRP, C-reactive protein; TNF, tumor necrosis factor; IL-1, -6, -10, -18, interleukin 1, 6, 10, 18; TNF-RI, RII, tumor necrosis factor receptor I, II; IL-1 RI, RII, interleukin 1 receptor antagonist I, II; sIL6-R, soluble form interleukin 6 receptor; mIL-6R, membrane-bound soluble interleukin 6 receptor.

Thus it is essential that the problem of managing patients with multiple injuries have to be shifted from early & effective resuscitation to the treatment of the host response to injury.

EFFECT OF OCCULT HYPOPERFUSION

The main principle in trauma care is to recognize & treat haemorrhage early, limiting the consequences of shock and hypoperfusion. Hypoperfusion remains still difficult to be diagnosed early as it favors adverse inflammatory and immunologic effects, coagulopathy, development of infection and organ failures, and finally precipitates late mortality(28). Patients with prolonged hypoperfusion have an increased risk of infection and mortality. Many studies established that patients with occult hypoperfusion (OH) at 24 hours had significantly higher ISS than patients without OH and those who got corrected by 12 hours, but no difference in patients who got corrected between 12 to 24 hours(29).

Occult hypoperfusion longer than 12 hours is a clear risk factor for the development of infections, with a subsequent increase in mortality, ICU days, length of stay and hospital costs(30). ISS and lactic acidemia correction at 12 hours are independently predictive of subsequent infections. This signifies the importance of correcting OH rapidly and suggests the importance of more aggressive treatment in attempts to improve LA to normal by 12 hours to minimize both mortality and infections(31).

SERUM LACTATE AND ITS SIGNIFICANCE IN POLYTRAUMA

Lactate production occurs in all tissues like brain, skeletal muscles, RBCs & kidneys even at baseline conditions under oxygen rich level. In normal conditions, lactate is rapidly cleared by liver metabolism & by reconversion of lactate to pyruvate. This helps in keeping blood lactate level less than 9 mg/dl. In an occult hypoperfusion state, anaerobic metabolism prevails wherein pyruvate is metabolized to lactate, finally producing less number of adenosine triphosphate (ATP) molecules (2 vs 36) than through the normal aerobic mechanism via TCA cycle. Persistent lactic acidosis may lead on to respiratory failure, multi organ dysfunction or death following major trauma(32). Lactic acidosis thus indicates occult or overt hypoperfusion.

Resuscitation in polytrauma patients has been traditionally guided by normalization of vital signs such as blood pressure, urine output & heart rate. However, these parameters have been proved to be inadequate in detecting the endpoint of resuscitation in critically ill patients. The ideal marker should be able to assess resolution of hypoperfusion. There have been few studies to date evaluating the significance of elevated blood lactate in detecting occult hypoperfusion. Persistent occult hypoperfusion has been proved to

be associated with increased morbidity & mortality and early correction seems to improve clinical outcome(28). There have been only very few studies assessing the prognostic value of blood lactate values on the outcome of high-risk, haemodynamically stable, trauma patients.

The field triage decision to transfer the trauma patient to a deputed trauma center should be systematic, rapid & accurate. At present, only heart rate, systolic blood pressure & urine output are used to decide transfer decisions, all of which are unreliable measures of acute haemorrhage. Systolic BP less than 90 mm Hg is still commonly used clinically to classify patients with shock, although ATLS suggests that relying solely on systolic BP to identify shock will result in delay in recognition of significant haemorrhage(33). Although we historically consider systolic BP of less than 90 mm Hg as shock, there is limited evidence to suggest that tissue hypoperfusion & ischemia are limited to patients with SBP< 90mmHg. Recent studies suggest that SBP of less than 110 mm Hg may more accurately reflect the first physiologic response of shock. Therefore, close monitoring of patients with SBP of 90-110 mmHg is needed to identify occult shock. But, expanding the trauma triage may lead on to over-triaging and extract the already resource-

strapped trauma centers. Here comes the usefulness of serum lactate and base deficit in recognizing patients with occult hypoperfusion or early shock secondary to haemorrhage(11).

Shock is responsible for inadequate oxygen delivery, resulting in tissue hypoxia, anaerobic metabolism, and lactate production. Lactate is thus a diagnostic and prognostic biomarker in sepsis and trauma. Lactic acidosis may persist despite controlling haemorrhage, due to flow demand mismatch, vasoconstriction, shock or other dysfunctional responses.

SIGNIFICANCE OF LACTATE CLEARANCE

Lactate clearance has recently been emerged as an important concept in haemorrhagic shock, as part of the quantitative resuscitation concept that aims to reach predefined physiologic goals to be achieved within the first hours. Studies show that poor lactate clearance is associated with increased mortality in septic shock(34). Jones et al have shown that attempting to normalize lactate clearance is not inferior to normalizing central venous oxygen saturation in septic shock(35). In septic patients, lactate clearance is measured over a prolonged time period (from 6 to 24 hrs), which may not be appropriate in trauma.

In trauma patients, there is a need for rapid assessment of resuscitation and of the diagnosis of occult hypoperfusion during its early phase. There is a need for early prognostic indicator that may identify patients at high risk of death. Lactate clearance can be a good prognostic indicator in assessing the outcome in a polytrauma patient(36). Abramson et al observed that lactate level were normalized in patients who recovered well, but not in cases with poor outcome(37).

INITIAL EVALUATION AND MANAGEMENT OF THE MULTIPLY INJURED PATIENT

The management of polytrauma patient is divided into pre-hospital and in-hospital phases(38). The chance of survival is dependent on immediate care following injury. The speed with which the lethal processes are identified and rectified determines the good outcome. Timely management is very important. Due to human imperfections, a standard protocol should be available. Rapid transport from the site of injury and proper prehospital care remains the mainstay of treatment. The standard ATLS protocol aids in good prehospital management. The initial priorities should be airway maintenance, breathing and circulation always.

The in-hospital period in the evaluation and management is divided into four different periods(39). This division allows anticipation of potential problems and sensible decisions.

Table-5 In-Hospital Periods in the Evaluation and Management of the Trauma Patient

1.	Acute “reanimation” period (1 to 3 hours)
2.	Primary “stabilization” period (1 to 48 hours)
3.	Secondary “regeneration” period (2 to 10 days)
4.	Tertiary “reconstruction and rehabilitation” period (weeks)

ACUTE REANIMATION PERIOD

This phase includes the time from initial admission to control of life threatening emergencies. Rapid systematic assessment is made to identify the potentially life threatening injuries followed by prioritized management of the airway and circulation. This is followed by secondary survey to identify other major injuries, which should be done only when there is no life threatening injuries.

PRIMARY STABILIZATION PERIOD

This period starts after stabilizing respiratory, haemodynamic and neurologic systems and start focusing on major extremity injuries, wherein temporary stabilization of the long bone fractures

are done with external fixator or splintage, arterial injuries are dealt with and compartments released. This period lasts up to 48 hours.

SECONDARY REGENERATION PERIOD

This is the phase, wherein the general condition of the patient is stabilized and monitored. It is vital to regularly reevaluate the constantly evolving clinical picture to avoid harmful impact of intensive treatment or the burden of complex surgical procedures. Physiologic and intensive care scoring systems may be used to monitor clinical progress. In the presence of systemic inflammation and MODS, appropriate supportive measures are taken in an intensive care unit.

TERTIARY RECONSTRUCTION AND REHABILITATION PERIOD

This phase includes the final reconstructive surgeries like fixing complex mid-face fractures, spinal, pelvic fractures and joint reconstruction. Only when adequate recovery been established should these surgeries be contemplated.

The use of predefined and validated algorithms effectively guides inexperienced personnel and reduces the mortality, especially of the moderately severe polytrauma patients (ISS between 20 and 50). The initial goal is to identify the major life threatening injuries

such as tension pneumothorax or haemothorax, cardiac tamponade, airway obstruction or injury causing asphyxia, open thoracic trauma or flail chest, and massive internal or external haemorrhage. The acute management of these patients may necessitate urgent transfer to the operating theatre to address life threatening issues without wasting time for the diagnostic algorithms and secondary survey. A pertinent example is to miss a major abdominal or pelvic haemorrhage and dealing with severe extremity injury. The treating team should be continuously alert to immediately address any unforeseen events. Continuous awareness of the team and flexibility to change the current management process are essential.

The continuous monitoring of the blood pressure, respiratory rate, pulse rate, saturation, temperature, electrocardiogram, insertion of urine and/or gastric catheters, acquisition of an initial full blood count, arterial blood gases and cross matching of the patient have been accepted as the gold standard line of initial management. Initial radiography includes chest anteroposterior, AP pelvis, and lateral cervical spine and use of FAST or abdominal ultra-sonogram.

VIEWS USED IN FAST SCANS

- Transverse subxiphoid view (pericardial effusion, left liver lobe)
- Right upper quadrant view (right liver lobe, right kidney, free fluid in Morrison's pouch)
- Longitudinal left upper quadrant view (spleen, left kidney, free fluid)
- Transverse and longitudinal suprapubic views (bladder, free fluid in Pouch of Douglas)
- Bilateral longitudinal thoracic views (pleural effusions)

The need for multi-slice whole body CT scan or total body digital radiography as a routine for all trauma patients is still under debate.

MANAGEMENT OF HAEMORRHAGIC SHOCK

Management of haemorrhage starts with pre-hospital phase by inserting IV cannula, preferably in ante-cubital fossa and start infusing fluids. Single internal jugular or subclavian vein lines are not preferred being too long and narrow to allow large amounts of fluid. Hence, a venous cut down may be done using the long

saphenous vein around the ankle. The choice of fluid still remains controversial. The infusion of a combination of crystalloid and blood at a 3:1 ratio is usually recommended. Colloids are also recommended as it maintains the intravascular space and reduces edema. This effect is enhanced when combined with hypertonic saline and dextran(40).

The frequent source of haemorrhage is usually from abdomen, thorax or pelvis. A high amount of clinical suspicion and rapid investigations like X-Rays and ultra-sonogram are essential.

An adequate clinical response includes improvement in pulse, blood pressure, capillary refill and urine output. In the severely injured patients, invasive monitoring like central venous or pulmonary artery pressure should be considered(41). Current goals aim at normalization of vitals and maintenance of central venous pressure between 8 and 15 mm Hg. Serial recording of acid base parameters, base excess and serum lactate will be useful in assessing the response to treatment and identifying occult hypoperfusion in apparently stable patients. Shock treatment is an ongoing process and surgical treatment should be considered if needed. Ongoing requirement of blood transfusion is decided by regular measurement of blood haemoglobin concentration. More recently, several methods

of improved monitoring of cardiovascular status have been introduced including gastric tonometry, near infrared spectroscopy, transthoracic impedance, echocardiography, central venous oximetry, and skeletal muscle acid-base estimation.

In case of emergency, O –ve blood which is the universal donor, should be given if cross matching is getting delayed. The need for infusion of blood products like packed cell, fresh frozen plasma or platelets are decided based on clinical judgment guided by laboratory results.

Haemorrhagic shock should be differentiated from cardiogenic and neurogenic shock before transfusing blood and fluids. A flat jugular vein indicates haemorrhagic shock, whereas a bulged jugular vein indicates cardiogenic shock, which might be due to cardiac tamponade, tension pneumothorax, and haemothorax or intraabdominal bleeding. These pathologies may need an emergency intervention like placement of a chest drain, pericardiocentesis or emergency thoracotomy. If there is indirect impairment of cardiac function, medical treatment should be introduced and normovolemia should be restored. A raised JVP in cardiogenic shock may be the result of right-sided heart failure. This should be confirmed by measurement of central venous pressure.

Table-6 Use of Preexisting Classification Systems to Assess Whether Patients Are Stable or Can Be Stabilized to Permit Definitive Fracture Fixation

	Parameter	Stable (Grade I)	Borderline (Grade II)	Unstable (Grade III)	In extremis (Grade IV)
Shock	Blood pressure (mm Hg)	100 or more	80 to 100	60 to 90	<50 to 60
	Blood units (2 hours)	0 to 2	2 to 8	5 to 15	>15
	Lactate levels	Normal range	Around 2.5 mmol/L	>2.5 mmol/L	Severe acidosis
	Base deficit (mmol/L)	Normal range	No data	No data	>6 to 8
	ATLS Classification	I	II to III	III to IV	IV
Coagulation	Platelet count (cells/µL)	>110,000	90,000 to 110,000	<70,000 to 90,000	<70,000
	Factor II and V (%)	90 to 100	70 to 80	50 to 70	<50
	Fibrinogen (g/dL)	>1	Around 1	<1	DIC
	D-dimer	Normal range	Abnormal	Abnormal	DIC
Temperature		>35°C	33° to 35°C	30° to 32°C	30° C or less

	Parameter	Stable (Grade I)	Borderline (Grade II)	Unstable (Grade III)	In extremis (Grade IV)
Soft tissue injuries	Lung function; PaO_2/FiO_2	350 to 400	300 to 350	200 to 300	<200
	Chest trauma scores; AIS	AIS I or II	AIS 2 or greater	AIS 2 or greater	AIS 3 or greater
	Chest trauma score; TTS	0	I to II	II to III	IV
	Abdominal trauma (Moore)	II or less	III or less	III	III or greater
	Pelvic trauma (AO class)	A type (AO)	B or C	C	C (crush, rollover abdomen)
	Extremities	AIS I to II	AIS II to III	AIS III to IV	Crush, rollover extremities

Three of the four categories must be met to allow classification into for a particular category. Patients who respond to resuscitation qualify for early definitive care as long as prolonged surgery is avoided(42).

STAGING OF PATIENT'S PHYSIOLOGICAL STATUS

Once the initial assessment and resuscitation are over, patient should be placed in one of four physiologic categories in order to plan subsequently. Any deterioration in clinical condition is identified early and proper measures taken. Achieving end points of resuscitation is of paramount importance to stratify the patients. End points of resuscitation include stable haemodynamics, stable oxygen saturation, lactate level less than 19.8 mg/dl, no coagulation disturbances, normal temperature, urinary output greater than 1 ml/kg/hr, and no requirement for inotropic support.

STABLE

Stable patients have no immediately life-threatening injuries, respond to initial therapy, and are haemodynamically stable without inotropic support. There is no evidence of physiologic disturbance such as coagulopathy or respiratory distress or ongoing occult hypoperfusion manifesting as abnormalities of acid-base status. They are not hypothermic. These patients have the physiologic reserve to withstand prolonged operative intervention where this is appropriate and can be managed using an early total care approach, with reconstruction of complex injuries.

BORDERLINE

Borderline patients can be stabilized in response to initial resuscitative attempts but have clinical features, or combinations of injury, which have been associated with poor outcome and put them at risk of rapid deterioration. These have been defined as follows:

- ISS greater than 40
- Hypothermia below 35° C
- Initial mean pulmonary arterial pressure greater than 24 mm Hg or a greater than 6 mm Hg rise in pulmonary artery pressure during intramedullary nailing or other operative intervention
- Multiple injuries (ISS greater than 20) in association with thoracic trauma (AIS greater than 2)
- Multiple injuries in association with severe abdominal or pelvic injury and haemorrhagic shock at presentation (systolic BP less than 90 mm Hg)
- Radiographic evidence of pulmonary contusion
- Patients with bilateral femoral fracture

- Patients with moderate or severe head injuries (AIS 3 or greater)

This group of patients can be initially managed using an early total care approach, but this should be undertaken with caution and forethought given to operative strategy should the patient require a rapid change of treatment rationale. Additional invasive monitoring should be instituted and provision made for ICU admission. A low threshold should be used for conversion to a damage control approach to management, as detailed later.

UNSTABLE

Patients who remain haemodynamically unstable despite initial intervention are at greatly increased risk of rapid deterioration, subsequent multiple organ failure, and death. Treatment in these cases has evolved to utilize a “damage control” approach. This entails rapid lifesaving surgery only if absolutely necessary and timely transfer to the intensive care unit for further stabilization and monitoring. Temporary stabilization of fractures using external fixation, haemorrhage control, and exteriorization of gastrointestinal injuries where possible is advocated. Complex reconstructive procedures should be delayed until stability is achieved and the acute immunoinflammatory response to injury has subsided. This

rationale is intended to reduce the magnitude of the “second hit” of operative intervention or at least delay it until the patient is physiologically equipped to cope.

IN EXTREMIS

These patients are very close to death, having suffered severe injuries, and often have ongoing uncontrolled blood loss. They remain severely unstable despite ongoing resuscitative efforts and are usually suffering from the effects of a “deadly triad” of hypothermia, acidosis, and coagulopathy(43). A damage control approach is certainly advocated. Only absolutely lifesaving procedures are attempted in order not to exhaust the biological reserve of these patients. The patients should then be transferred directly to intensive care for invasive monitoring and advanced haematologic, pulmonary, and cardiovascular support. Orthopaedic injuries can be stabilized rapidly in the emergency department or ICU using external fixation and this should not delay other therapy. Further reconstructive surgery is delayed and can be performed if the patient survives.

SURGICAL PRIORITIES FOR LIFE SAVING SURGERIES

In polytrauma patients, correct decision making regarding requirement of any emergent operative treatment should be made.

Cardiac tamponade, arterial injuries to major blood vessels, and head trauma with imminent incarceration are some of the emergent conditions requiring urgent treatment that does not permit the use of prolonged diagnostic procedures.

HAEMOTHORAX

Haemothorax is usually diagnosed easily in a chest x ray. If extensive lung contusion or atelectasis is present, USG or CT might be helpful. Significant bleeding into the pleural space with a resultant haemothorax is treated by intercostal drainage. It is inserted in 5^{th} intercostal space in mid-axillary line. Lower insertion risks injury to diaphragm or intra-abdominal organs. Blunt dissection is carried to avoid injury, even while the operator is confident, because intra-abdominal injuries may lead to increased intra-abdominal pressure and diaphragmatic elevation or rupture. A traditional tube of size 28Fr is used. Large diameter tube should be used to allow rapid evacuation so that the drained contents can be taken as representative of total thoracic blood loss. Direct the tube caudally for haemothorax and cephaladly for pneumothorax. Indications for emergency thoracotomy are traumatic arrest and recalcitrant profound hypotension in penetrating trauma, rapid exsanguination with more than 1500 ml of blood initially or 250

ml/hr after chest tube insertion and unresponsive hypotension in blunt thoracic trauma.

MEDIASTINAL HAEMORRHAGE AND THORACIC AORTIC INJURY

Mediastinal haemorrhage can be erroneously diagnosed with chest x ray. In the presence of dilated jugular veins, contrast enhanced CT or Angiography can diagnose the condition. In most cases of thoracic aorta rupture who manage to come alive to the ER, the adventitia is usually preserved and further intrathoracic blood loss is prevented by the parietal pleura. Repair can be delayed in the presence of other life threatening injuries and sometimes, conservative management may be successful. Emergency surgery is indicated during unexplained haemodynamic instability, haemorrhage of more than 500 ml/hr in ICD tube, a blood pressure gradient of >30 mmHg between upper & lower limbs.

SEVERE PELVIC TRAUMA: APPLICATION OF SHEETS, BINDERS, EXTERNAL FIXATORS AND PACKING

Pelvic fracture is often seen in conjunction with major trauma and can lead to rapid occult haemorrhage. Bleeding is more common from multiple small vessels rather than from injured major vessels and, because of the large volume of the retroperitoneum, spontaneous arrest of the bleeding is unlikely(44). Treatment with

pneumatic anti-shock garment or pelvic belt-straps can give some temporary stabilization. Embolization of blood vessels may be recommended in continued arterial haemorrhage. Otherwise, use of an external fixator, pelvic C-clamp and open tamponade by packing is recommended in routine cases(45). In a C-type injury with vertical pelvic instability, the lower extremity of the appropriate side should be accessible for reduction. If there is intraperitoneal fluid, midline laparotomy with pelvic external fixation is done. If there is no evidence of intraabdominal fluid but a major pelvic haemorrhage is suspected, a lower midline laparotomy is suggested. Large bleeding vessels are ligated and for diffuse bleeding, well-directed packing with external fixation is recommended. If the haemorrhage is obviously from a deep dorsal source, like in cases of posterior pelvic instability, attempts at further extraperitoneal exploration should be made in the presacral region. Large bleeding vessels are identified and addressed. In ongoing shock, re-intervention is planned.

EARLY TOTAL CARE VS DAMAGE CONTROL ORTHOPAEDICS

Before fracture fixation was routinely performed, patients fared very badly due to complications like fat embolism and organ failure. Pulmonary dysfunction is the hallmark of this syndrome. Fat

embolism was found to be caused by release of intramedullary contents and fat into the circulation from an unstabilized fracture. Hence, it was planned to operate on all femur fractures as early as possible. Many authors found increased clinical outcome, less number of pulmonary dysfunction, pneumonia and ARDS, shorter stay in ICU and better survival after femur fixation was started. This is now referred to as early total care as proposed by Bone et al(46). Patients with delayed fracture fixation had a longer course in ICU and hospital. It is therefore accepted that the major aim of treating polytrauma patients are early stabilization of fractures, the main pre-requisite being optimum retrieval time and condition. With improved cardiovascular monitoring and facilities and ICU facilities, a more aggressive surgical approach is nowadays possible.

The strict application of these rules do not apply in patients with high ISS, brain injury or severe chest trauma, who are at risk for a poor late outcome. The concept of damage control provides solution to these kinds of borderline patients(47). The term damage control was originally used by the US navy as “the capacity of the ship to absorb damage and maintain mission integrity”. In the polytraumatized patients, surgical treatment is aimed to control but not definitely treat the injuries early after trauma. After restoration

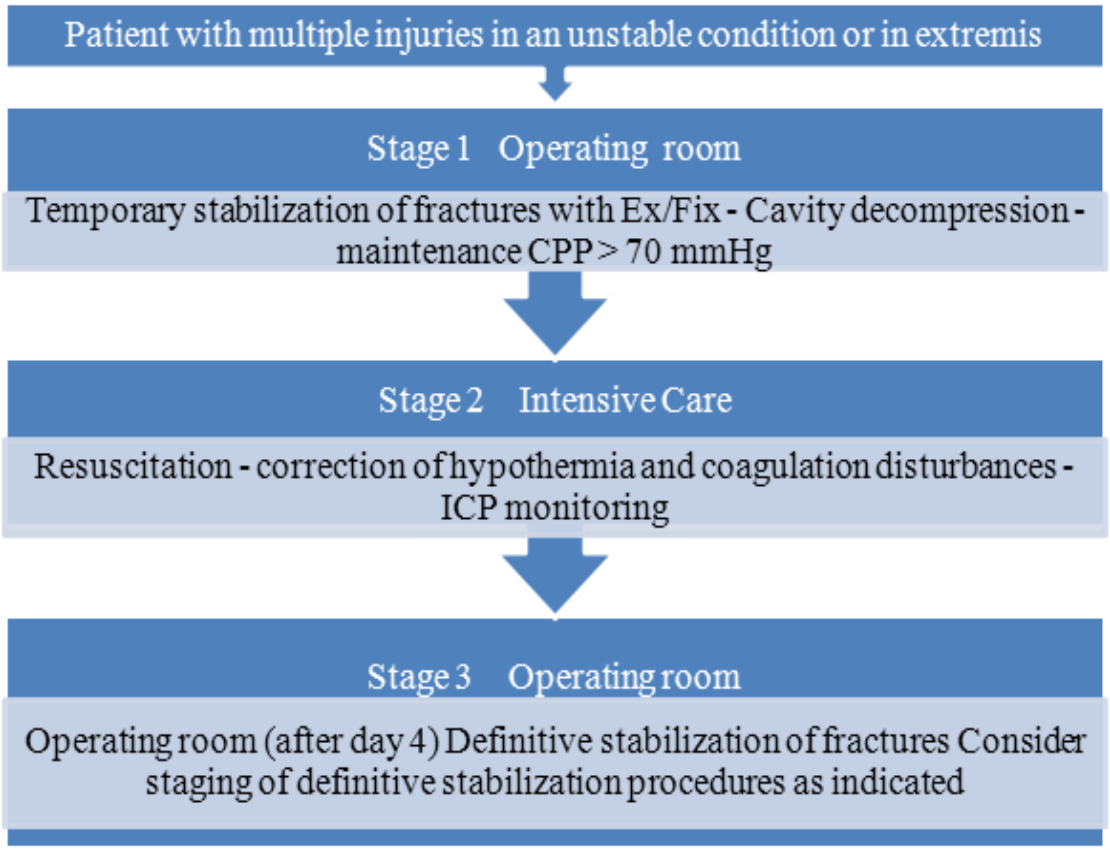
of normal physiology like core temperature, haemodynamics, coagulation, respiratory status, the definitive management of injuries is planned(48). Thus, the damage control concept consists of three different components: resuscitative surgery for rapid haemorrhage control, restoration of normal physiologic parameters and definitive surgical management(49).

In the damage control concept, the first stage involves early temporary stabilization of the fractured pelvis or any long bone, the most popular tool of a trauma surgeon for this purpose being an external fixator(49). The second stage involves resuscitation in the ICU with optimization of the patient's haemodynamic status. The third stage involves delayed definitive fracture management when the patient's condition allows.

In patients with head, chest or pelvic injury, early fixation of associated long bone fracture is debatable as an acute change in the clinical condition may occur. The practice of delaying the definitive surgery in damage control orthopaedics aims to reduce the biologic load of surgical trauma in the already traumatized patient. This hypothesis was proved in a randomized controlled trial by measuring pro-inflammatory cytokines. Clinically stable polytrauma patients with ISS > 16 with a femoral fracture were randomized for ETC by

primary femoral nailing and DCO by initial external fixation followed by delayed nailing. A sustained inflammatory response (higher levels of IL-6) was measured after primary IL nailing, but not after secondary nailing(50). The authors concluded that DCO minimizes the additional surgical impact induced by acute stabilization of the fracture(50).

THE STAGING OF DAMAGE CONTROL ORTHOPAEDICS



INDICATIONS FOR EARLY TOTAL CARE

- Stable haemodynamics
- No need for vasoactive/inotropic stimulation
- No hypoxemia, no hypercapnia
- Lactate less than 2 mmol/L
- Normal coagulation
- Normothermia
- Urinary output greater than 1 mL/kg/hr

INDICATIONS FOR DAMAGE CONTROL SURGERY

- Physiological criteria – hypothermia, coagulopathy, acidosis = lethal triad
- Complex pattern of severe injuries with probable major blood loss

UNSTABLE PELVIC INJURIES

The management of unstable pelvic injuries will be easier if a standardized protocol is used. A simple ABC system of classification can assist in decision making process(45). Type A injuries include avulsion fractures, pelvic rim and undisplaced

anterior pelvic ring fractures. The posterior rim is stable. Type B injuries comprise fractures with only partially intact posterior structures and rotational dislocations may be possible. Internal rotational dislocation may carry a high risk of intra-abdominal injuries. In external rotation type of injuries, urogenital lesions and haemorrhagic complications are more common. A CT scan or diagonal inlet and outlet view is needed to differentiate type B from type C injuries. In type C, pelvis shows translational instability of the dorsal pelvic ring, such that the stabilizing structures are all divided. This is associated with high risk of haemorrhage.

Type A injuries need no treatment. Type B injuries need osteosynthesis of anterior pelvic ring only. Type C injuries need anterior and posterior osteosynthesis for adequate stability.

Complex pelvic injuries are those with associated injury to any local pelvic organs. The primary aim of management in these kinds of injuries is a combined strategy of intensive shock treatment, early stabilization of the pelvic ring and potential operative haemorrhage control and packing. Urogenital injuries are managed with primary repair for intraperitoneal bladder rupture, and suprapubic catheterization or transurethral splinting and late repair for urethral injuries. A temporary colostomy is done for injury to rectum or anus.

INTENSIVE CARE MANAGEMENT

Ventilation strategies

Multiple trauma patients often present with thoracic trauma and suffer from a variable degree of respiratory insufficiency. Management strategies should begin on arrival at the trauma center. The objective is to minimize the risk of development of atelectasis and/or parenchymal damage. Mechanical ventilation should facilitate alveolar recruitment and enhance intra-pulmonary gas distribution. Modern ventilation strategies with low tidal volume (4 to 8 ml/kg), suitable positive end-expiratory pressure (PEEP), low airway pressures (less than 35cm H_2O) and an inspiratory oxygen concentration of 55 to 60% are ideal. Permissive hypercapnia (PHC) may be allowed up to a certain degree(51). Clinical experience shows that the pressure controlled ventilation with inversed ratio ventilation [I:E (1:1 to4:1)], low tidal volumes, frequencies of 10 to 15/min, PHC (pCO_2 about 70 mm Hg), and an individual PEEP (5 to 12 cm H_2O), a high oxygen concentration (F_iO_2 less than 0.5) and a high airway pressure can prevent the lung from further ventilation damage(52).

ADULT RESPIRATORY DISTRESS SYNDROME

ARDS can be caused by severe pneumonia or trauma. In ARDS, lungs become swollen with water and protein and breathing

becomes impossible leading to death in 3 to 40% of cases. Activated blood cells, cytokines, toxins, cell debris, and local tissue damage facilitate endothelial cell damage leading to decompensation of lymph drainage and pulmonary interstitial edema. Patients with ARDS have higher hospital mortality rates and reduced long term lung function and quality of life. ARDS is treated with mechanical ventilation. Low to moderate dose steroid may be tried. The formation of scar tissue is often the result of high intra-alveolar pressures because of inadequate ventilation techniques.

MULTIPLE ORGAN DYSFUNCTION SYNDROME

MODS is the result of an inappropriate generalized inflammatory response of the host to a variety of insults. In the early phase, circulating cytokines are the culprit, whereas in the late stage, inflammatory mediators in interstitial space of various organs are considered a main mechanism of parenchymal injury. The sequence and severity of damage depends on the difference in constitutive expression and the up regulation of adhesion molecules in vascular beds and the density and potency of intrinsic inflammatory cells in different organs. The most commonly reported sequence of organ damage is lung, followed by liver and intestine(53).

AIM OF STUDY

Primary aim of the study is to study the significance of blood parameters at the time of admission in predicting the morbidity & mortality of multiply injured patients.

To understand the significance of blood lactate and lactate clearance in polytrauma and compound injury patients. We aim to answer the following questions. 1) Are serum lactate and lactate clearance independent and additional predictors of outcome in polytrauma patients? 2) How do these two variables evolve during the initials hours of resuscitation and change according to the effectiveness of resuscitation? 3) Do these variables predict any other clinically relevant endpoints beside mortality? 4) Do these values correlate with pre-hospital care, injury-admission interval, vital parameters and trauma scoring system? 5) Do these variables predict outcome in the high risk, but haemodynamically stable patients?

The extension of this study is to explore the role of serum lactate in predicting timing of definitive and reconstructive procedures in polytrauma and compound fractures.

MATERIALS AND METHODS

This was a prospective observational and analytical study conducted in our institute of Orthopaedics & Traumatology, Rajiv Gandhi Govt. General Hospital, Chennai from the period of March 2015 to September 2015 with approval from Institution's Ethics Committee. We selected 69 patients of multiply injured and compound injury patients admitted in our emergency trauma ward. Patients for my study were selected using the inclusion and exclusion criteria formed.

INCLUSION CRITERIA

- All compound fractures of lower limbs
- Polytrauma patients with ISS more than 16
- Both male & female of age above 14
- Trauma patients with hypovolemic shock on arrival

EXCLUSION CRITERIA

- Closed isolated long bone and/or small bone fractures
- Isolated ankle & foot compound injuries
- Associated spinal cord injury with neurological deficit

❖ Patients with H/o taking immunosuppressive/ ART drugs

Patient was first received in the emergency ward and vital parameters, namely heart rate, systolic BP, mean arterial pressure, urine output, SPO2, GCS, respiratory rate and temperature were measured and recorded along with other information including demographic data. The prehospital time was noted, which is the time interval between time of injury and arrival to the hospital. Details of pre-hospital care given were recorded. Then resuscitation was started according to standard ATLS protocol. Abbreviated Injury Score of all external injuries and total New Injury Severity Score were calculated. Mangled Extremity Severity Score and Ganga Hospital Open Injury Score were calculated for all compound fractures. Blood collected and sent for routine investigations, blood grouping and cross matching and also for serum lactate measurement. Blood transfusion was planned according to the clinical observation and Hb and PCV values. Once the patient got stabilized, he was shifted for investigations like X rays, CT scan, USG, ECG, etc... Any immediate lifesaving procedures like intercostal drainage, thoracocentesis, needle thoracotomy, pelvic external fixator, fracture splintage were done in the emergency ward itself. The decision regarding implementation of damage control orthopaedics vs early total care was made according to the existing

guidelines. All compound grade 2 & 3 fractures were taken for emergency wound debridement and external fixator application. The comorbidities were simultaneously found out, necessary specialists' opinions obtained and interventions given as needed. All vital parameters were continuously monitored. Serum lactate was again measured at 6 hours and then at 24 hours. Blood sugar, urea, creatinine, complete blood count, serum electrolytes were measured again after 24 hrs. Intensive monitoring and care were given till all vital parameters became normal. Oxygen support was given when pulse-oximeter showed reduced oxygen saturation in blood and stopped when it rose to more than 95%. Endotracheal intubation and ventilator support were given with the help of duty anaesthetists in case of acute respiratory distress or respiratory arrest. Inotropes were sometimes given with the advice of intensivists. Any complications were identified early and managed quickly. Once the patients' vitals had become normal and considered free from all complications, they were transferred to the wards and definitive treatment planned.

ENDPOINTS

The final outcome of the patient was measured qualitatively as follows.

1) Early death of the patient, defined as death within 48 hrs,

2) ICU length of stay more than 2 days

3) Death within 30 days/ complications like sepsis, MODS,etc.

4) Recovery with sequelae/ Late recovery

5) Full early recovery

Apart from this, the requirement for an emergency procedure like intercostal drainage, emergency surgery, emergency embolization, or emergency transfusions were recorded.

METHOD OF SERUM LACTATE MEASUREMENT

Serum lactate measurement is used to assess acid-base status of the blood and is done to diagnose lactic acidosis and thus hypoperfusion and shock. Enzymatic methods are now preferred to calorimetric and titrimetric methods as it is simple, accurate, specific and reproducible. The first enzymatic method was based on transferring hydrogen ion from lactate to potassium ferricyanide by lactate dehydrogenase which is cumbersome. Subsequent methods involved the UV measurement of the formation of NADH. In 1974, Gutman and Wahlefeld described a method that measures NADH formed from lactate by LD, using hydrazine as a trapping agent for pyruvate. A method described by Noll is also based on the catalytic action of LD but uses ALT to more rapidly remove the pyruvate. The

method done in this study uses an enzymatic reaction to convert lactate to pyruvate. The hydrogen peroxide produced by this enzymatic reaction is then used in another reaction to generate a colored dye. This method offers longer reagent stability than the previously described methods.

TEST PRINCIPLE

Calorimetric assay

L-Lactate is oxidized to pyruvate by the enzyme lactate oxidase (LOD). The hydrogen peroxide formed as a byproduct is then generated into a colored dye by peroxidase enzyme (POD).

$$\text{L-Lactate} + O_2 \xrightarrow{\text{(LOD)}} \text{Pyruvate} + H_2O_2$$

$$2\,H_2O_2 + \text{H Donor} + 4\,\text{AAP} \xrightarrow{\text{(POD)}} \text{Chromogen} + 2H_2O$$

The intensity of the color is directly proportional to the L-Lactate concentration. It is determined by measuring the increase in absorbance.

Blood specimens are collected in sodium fluoride (Na F) coated blood collection tubes and stored in ice box. The sample is then transported to the biochemistry laboratory and centrifuged within 15 mins. Serum lactate concentration is then measured with

above said method using auto analyzer. The level is measured in mg/dl and can be converted to mmol/l using the conversion factors.

mmol/l × 9.009 = mg/dl

mmol/l × 90.09 = mg/l

mg/dl × 0.111 = mmol/l

The coefficients of variation of the measurement of blood lactate were 3.8% (at 1.15mm/l) and 2.6% (at 4.5 mm/l) in our laboratory. We analyzed previous studies and observed that the threshold for abnormal values was 19.8 mg/dl (mean and median extremes being 18 and 21.5 mg/dl). Thus, the normal range is considered as 19.8 mg/dl or lower.

Lactate clearance is then calculated using the formula-

$$\text{Lactate clearance} = \frac{(\text{Lactate initial} - \text{Lactate delayed})}{\text{Lactate initial}} \times 100$$

The lactate clearance is calculated at 0 – 6 hrs, 0 – 24 hrs and then at 6 – 24 hrs.

STATISTICAL ANALYSIS

Serum lactate and lactate clearances of all patients are measured and charted along with all parameters. Outcome variables are also recorded in defined manner. Mean and standard deviations were calculated for each variable. Comparison of two means was performed using unpaired student t test, comparison of two medians was performed using Mann – Whitney test, comparison of proportions was performed using fisher exact method. Correlation between two variables was assessed using linear regression analysis.

Multiple logistic regressions were performed to assess the role of serum lactate and lactate clearance. We included initial lactate level, lactate clearances and ISS scores. Discrimination of the final models was assessed by measurement of area under receiver operating characteristic curve and their calibration by Hosmer- Lemeshow statistic. Odds ratios and their 95 % confidence interval were calculated.

RESULTS

We analyzed a total of 69 patients of mean age 40.61 years. As ours is a tertiary center and most of the patients are being referred from a nearby taluk or district headquarters hospital and also from various medical colleges, we received the patients after an average delay of 5.74 hours from the injury time. The patients those we analyzed were of average Injury Severity Score 20.72. The compound injury patients were of average mangled extremity severity score 4.18. The average SBP of the polytrauma patients we received was 99.42 and heart rate was 100.95, thus the average shock index being nearly 1. The average serum lactate at the time of admission was 35.08 mg/dl and lactate clearance in the first 24 hours was 15.181. Serum lactate was elevated in 67 patients.

Age distribution and its effect on final outcome are described in the following table.

TABLE – 7 AGE DISTRIBUTION

Age in Years/ Outcome	< 30 Yrs	31-40 Yrs	41-50 Yrs	51-60 Yrs	>60 Yrs	Total
Early Recovery	9	12	4	4	0	29
Late Recovery	8	2	2	5	5	22
Recovered with sequelae	6	2	1	3	2	14
Death	1	0	2	1	0	4
Total	24	16	9	13	7	69

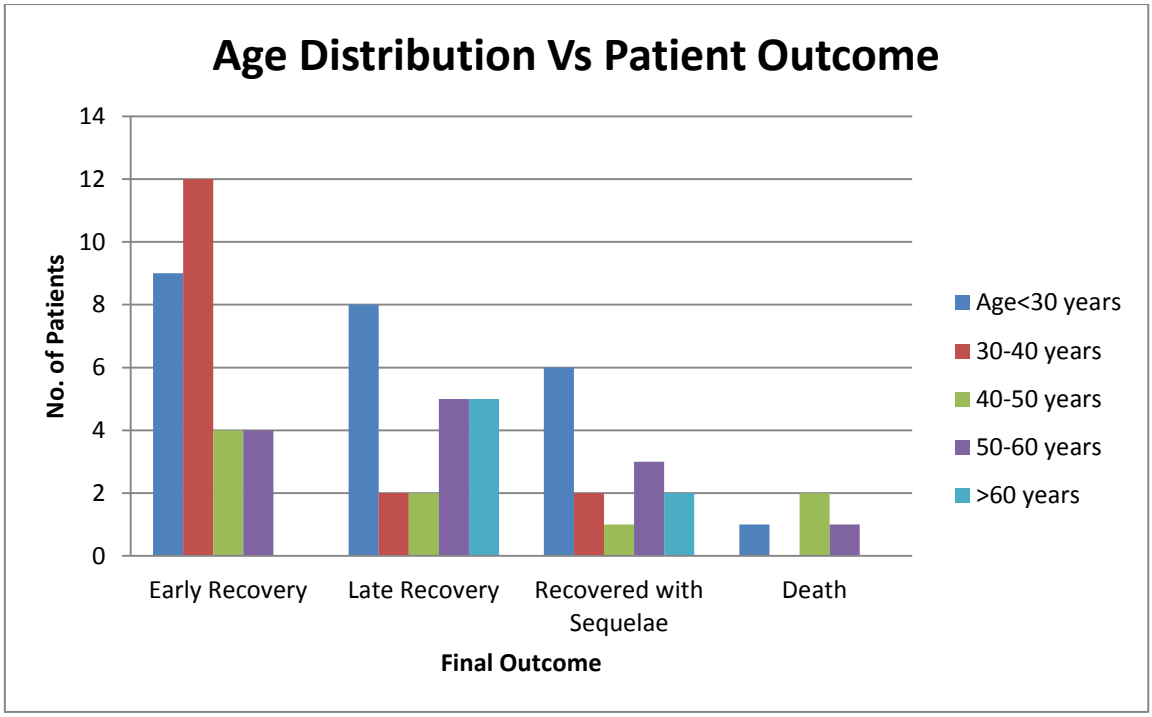
As described in the above table, out of all the patients we analyzed, 36% of the patients fall under 30 years age group, 23% from 31 to 40 years age group, 13% from 41-50 years, 18% from 51 to 60 years and 11% over 60 years. Better outcome is seen in age groups less than 30 (32%) and 31-40 (44%). On applying Pearson Chi-square test, it was found that there was not much significant correlation (p value 0.079) between age and outcome.

TABLE – 8 SEX DISTRIBUTION

Outcome	Male	Female	Total
Early Recovery	29	0	29
Late recovery	19	3	22
Recovered with sequelae	14	0	14
Death	3	1	4
Total	65	4	69

In our study, we found 95% of the patients were male. We found not much significance with sex and outcome (p value 0.039).

In our study, there were totally 65 male and 4 female patients. One female patient died of septicemia and sudden cardiorespiratory arrest after 2 days of wound debridement and tibial external fixation for comp GrIIIB Both Bones fracture with high contamination.


Age Distribution Vs Patient Outcome
No. of Patients
14
12
10
8
6
4
2
0
Early Recovery
Late Recovery
Recovered with Sequelae
Death
Final Outcome
Age<30 years
30-40 years
40-50 years
50-60 years
>60 years

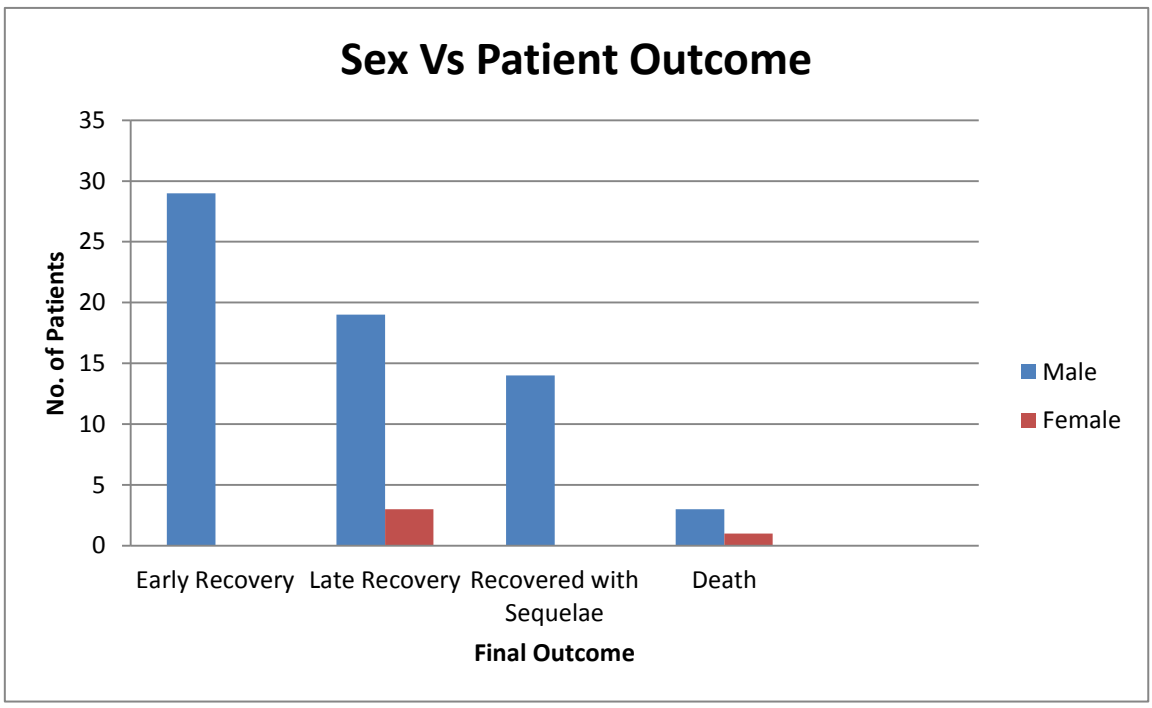

Sex Vs Patient Outcome
No. of Patients
35
30
25
20
15
10
5
0
Early Recovery
Late Recovery
Recovered with Sequelae
Death
Final Outcome
Male
Female

TABLE – 9 MECHANISM OF INJURY VS OUTCOME

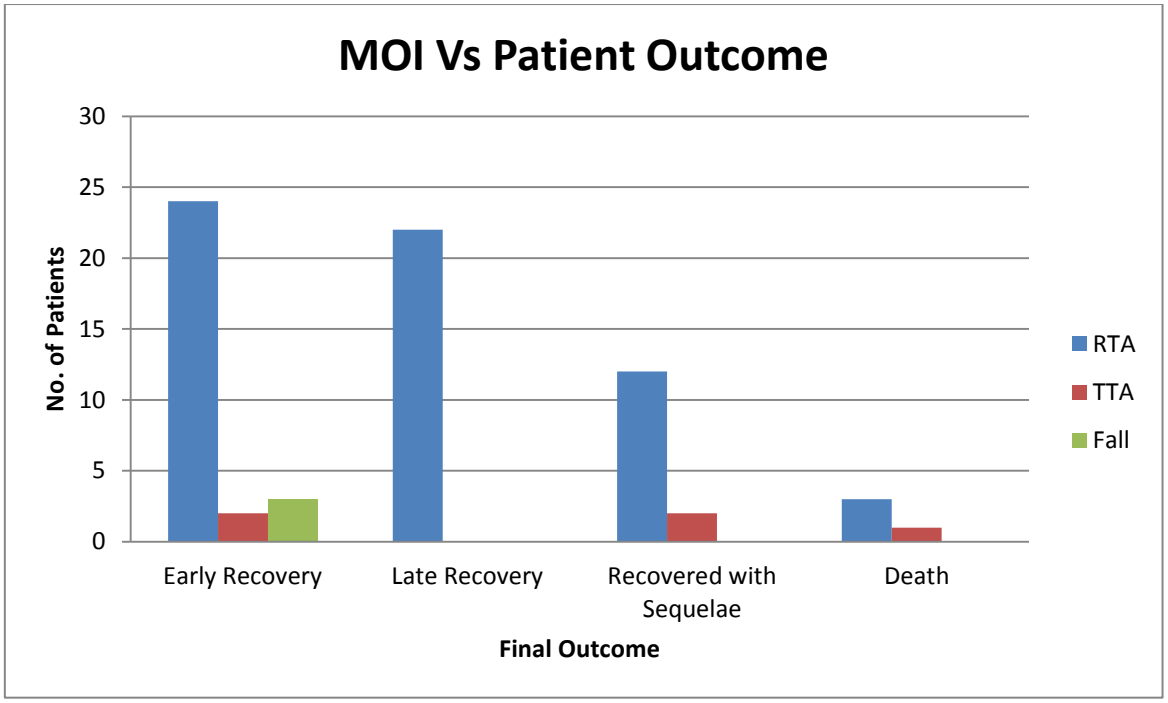
MOI/ Outcome	RTA	TTA	Fall	Total
Early Recovery	24	2	3	29
Late Recovery	22	0	0	22
Recovered with sequelae	12	2	0	14
Death	3	1	0	4
Total	61	5	3	69

According to our study, around 92% of patients analyzed under our inclusion criteria had sustained a road traffic accident. 5% sustained train traffic accident and 3% had fall from height. We observed 37% of RTA patients to have recovered early, whereas 40% recovered early in a TTA and all three patients who fell from height recovered early. There was no statistically significant correlation between mechanism of injury and final outcome (p value 0.519).

TABLE 10 LENGTH OF ICU STAY

ICU Stay/ outcome	No Stay	<12 hrs	13-24hrs	25-48hrs	>48hrs	Total
Early Recovery	3	9	13	4	0	29
Late Recovery	0	2	5	10	5	22
Recovered with sequelae	0	0	2	6	6	14
Death	0	2	0	1	1	4
Total	3	13	20	21	12	69

Around 5% of the patients did not need any ICU stay. 15% of the patients stayed in ICU for less than 12 hours. 30 % of the patients stayed for 13-24 hours, 32% for 25-48 hours and 18 % for more than 48 hours. All three patients who did not need ICU stay recovered very well. Of the 13 patients, who stayed for less than 12 hours in ICU, 2 patients died within that period. Hence, we found no statistically significant correlation between ICU stay and outcome (p value 0.029).


MOI Vs Patient Outcome
30
25
20
15
10
5
0
No. of Patients
Early Recovery
Late Recovery
Recovered with Sequelae
Death
Final Outcome
RTA
TTA
Fall

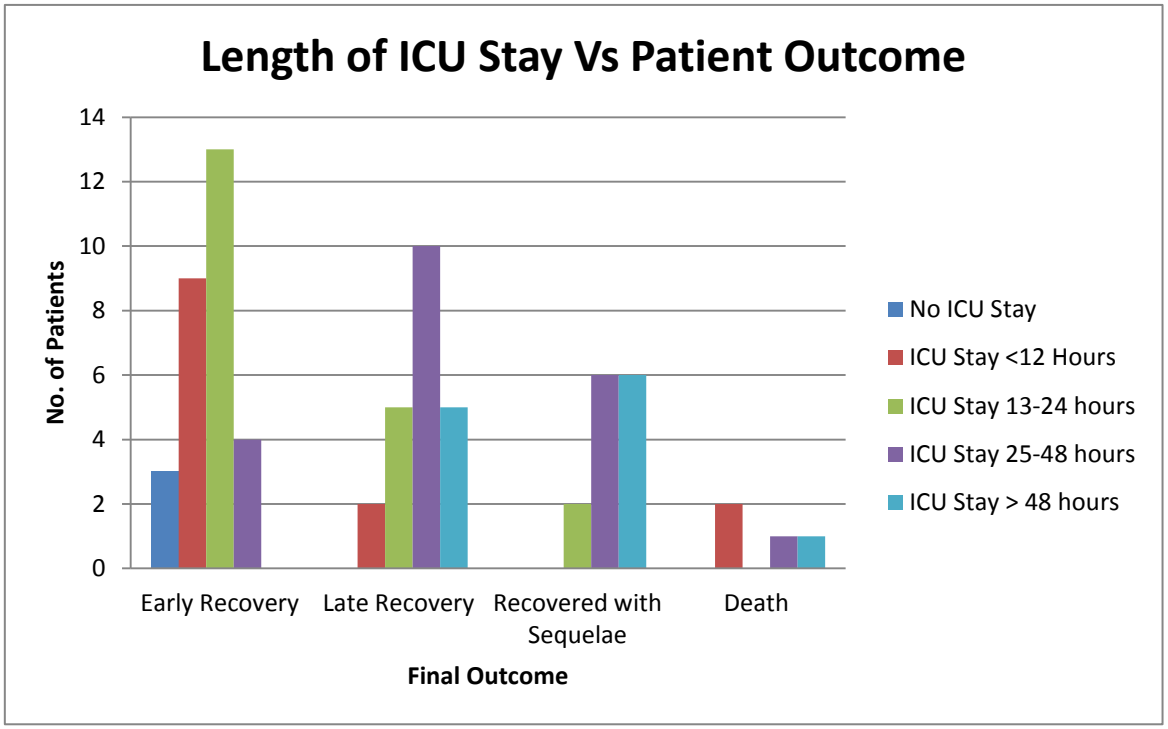

Length of ICU Stay Vs Patient Outcome
14
12
10
8
6
4
2
0
No. of Patients
Early Recovery
Late Recovery
Recovered with Sequelae
Death
Final Outcome
No ICU Stay
ICU Stay <12 Hours
ICU Stay 13-24 hours
ICU Stay 25-48 hours
ICU Stay > 48 hours

TABLE 11 ISS VS OUTCOME

Outcome	No. of Patients	Mean ISS	Minimum ISS	Maximum ISS
Early recovery	29	15.20	9	25
Late recovery	22	18.65	9	32
Recovered with sequelae	14	29.69	16	50
Death	4	41.67	16	75
Total	69	20.72	9	75

The above table signifies the association of injury severity score and the final outcome analyzed using ANOVA method (p value 0.001). The mean ISS for the patients who died is 41.67 when compared to mean ISS of patients who recovered early, which is 15.20.

TABLE 12 SBP vs OUTCOME

Outcome	No. of Patients	Mean SBP	Minimum SBP	Maximum SBP
Early recovery	25	103.84	90	130
Late recovery	21	98.48	90	124
Recovered with sequelae	13	94.46	86	110
Death	3	90.67	80	98
Total	62	99.42	80	130

The above table describes the association of systolic BP at the time of admission in predicting the final outcome. The table shows decreased SBP at the time of admission results in poor outcome (p value 0.003). In the above table, it is seen that the patients who recovered early, had a mean systolic BP of 103.8 at the time of admission. Patients who recovered late had a mean BP of 98.48, who recovered with sequelae had 94.46 and who died had a mean BP of 90.67. Thus we found a statistically significant correlation between systolic BP at the time of admission and the final outcome (p value 0.003).

TABLE 13 SERUM LACTATE VS FINAL OUTCOME

	Outcome	No. of patients	Mean Serum Lactate	Minimum Serum Lactate	Maximum Serum Lactate
Sr Lactate mg/dl – on admission	Early recovery	29	25.61	17	35
	Late recovery	22	34.95	25	45
	Recovered with sequelae	14	44.22	24	70
	Death	4	75.27	68	85
	Total	69	35.08	17	85
Sr Lactate mg/dl - after 24 hrs of admission	Early recovery	29	19.13	15	22
	Late recovery	22	24.88	19	33
	Recovered with sequelae	14	33.00	20	65
	Death	4	61.20	58	64
	Total	69	25.44	15	65

In our study, we observed that the patients who recovered early had a mean serum lactate value of 25.61mg/dl at the time of admission and got their lactate value corrected at 24 hours after admission. Patients who recovered late had mean lactate value of 34.95mg/dl at admission and 24.88 mg/dl at 24 hours. Patients who

recovered with sequelae had 44.22mg/dl at admission and 33mg/dl at 24 hours. Those who died had 75.27mg/dl at admission and 61.2mg/dl at 24 hours.

We can see clearly that increased lactate at the time of admission and persistent elevation of lactate at 24 hours resulted in very poor outcome (p value 0.0001 – highly significant). In one patient with compound GrIIIB both bones leg fracture, whose injury severity score was just 16 at the time of admission and systolic BP of 98 mmHg at the time of admission predicted very good outcome as we relied only on these parameters. But the serum lactate of this patient was very much elevated which would have predicted the ongoing septicemia and crush syndrome if relied on lactate values for resuscitation and thus would have avoided the death of the patient.

TABLE 14 LACTATE CLEARANCE VS FINAL OUTCOME

Outcome	No. of Patients	Mean Lactate Clearance%	Minimum Lactate Clearance%	Maximum Lactate Clearance%
Early recovery	29	13.023	-1.2	27.1
Late recovery	22	15.602	6.5	26.2
Recovered with sequelae	14	13.085	5.3	37.8
Death	4	2.910	-3.8	6.8
Total	69	13.017	-3.8	37.8

The significance of lactate clearance in predicting the final outcome was analyzed by ANOVA and post hoc hypothesis was subsequently formulated, which illustrated a significant association with p value of 0.025 (significant).The mean lactate clearance (0-24 hours) was calculated and observed that the patients who recovered early had a mean lactate clearance of 13.023%, whereas the patients who recovered late had 15.6, who recovered with sequelae had 13.09 and who died had mean lactate clearance of only 2.9%. We found a statistically significant association between poor lactate clearance and bad outcome in polytrauma patients.

TABLE 15 ISS VS SERUM LACTATE

The correlation of Injury severity score and serum lactate at admission is checked which shows a significant correlation at 0.001 level (2-tailed).

		ISS	**Sr Lactate mg/dl**
ISS	Pearson Correlation	1	0.807
	Sig. (2-tailed)	0	0.001
	N	69	69
Sr Lactate mg/dl	Pearson Correlation	0.807	1
	Sig. (2-tailed)	0.001	0
	N	69	69

Our study shows a significant positive correlation between injury severity score and serum lactate level at the time of admission.

TABLE 16 SBP VS SERUM LACTATE

		Sr Lactate mg/dl	SBP mm Hg
Sr Lactate mg/dl	Pearson Correlation	1	-0.569
	Sig. (2-tailed)	0	0.001
	N	69	69
SBP mm Hg	Pearson Correlation	-0.569	1
	Sig. (2-tailed)	0.001	0
	N	69	69

The correlation of systolic BP and serum Lactate at the time of admission was checked which also showed a significant correlation at 0.001 level (2-tailed).

The correlation study was also done individually between serum lactate at admission and amount of blood transfusions needed, length of ICU stay needed for the patient, any complications that the patient have faced and also the need of emergency procedures within 24 hours of admission which also gave mildly significant correlation, probably because of the relatively smaller sample size.

TABLE 17 SERUM LACTATE VS LENGTH OF ICU STAY

		Sr Lactate mg/dl –on admission	**Length of ICU stay in hours**
Sr Lactate mg/dl – on admission	Pearson Correlation	1	0.752
	Sig. (2-tailed)	0	0.001
	N	69	66
Length of ICU stay in hours	Pearson Correlation	0.752	1
	Sig. (2-tailed)	0.001	0
	N	66	66

TABLE 18 SERUM LACTATE VS NEED FOR BLOOD TRANSFUSION

		Sr Lactate mg/dl - on admission	**Blood transfused in units**
Sr Lactate mg/dl – on admission	Pearson Correlation	1	0.687
	Sig. (2-tailed)	0	0.001
	N	69	68
Blood transfused in units	Pearson Correlation	0.687	1
	Sig. (2-tailed)	0.001	0
	N	68	68

DISCUSSION

In our study, we have found the following significant findings: Most of the polytrauma patients we analyzed are of very young age group. Around 36% of the patients belong to age group less than 30 years, 23 % belong to 21-30 years, 13 % belong to 41-50 years, 18 % belong to 51 – 60 years and 12 % more than 60 years. Literature also supports this fact and the reason might be due to increased road traffic accidents affecting young active individuals. Also we observed relatively good outcome in young patients, which could be due to better endurance and less co-morbidities.

Of the total 69 patients we took for study, only 4 were female and all others were male. Of the total 4 mortalities, 1 was a female and others were male. 62 out of 69 patients had sustained injury due to a road traffic accident, 4 patients had train traffic accident and 3 patients had a history of fall from height. 3 patients of RTA and 1 patient of TTA died.

Of the total 69 patients, 13 patients required less than 12 hours of ICU stay and they all have recovered early except two patients who died before this period in ICU itself. There was a significant correlation between Injury Severity Score and final outcome shown

in our study with p value of 0.001. But one patient with ISS of 16, who got compound GrIIIB BB fractures of leg, fared well with external fixator on day 1 and suddenly succumbed to septicemia and ARDS on day 3. Hence, it shows that trauma scoring per se cannot predict outcome in all cases. The systolic BP at the time of admission correlates well with final outcome signified by a p value of 0.003. Literature says that shock index, which is a ratio of heart rate to systolic BP is a better predictor of outcome than systolic BP alone.

And lastly, we found that serum lactate values and lactate clearance in the first 24 hours correlates well with the final outcome. Patients who recovered early had a mean admission lactate value of 25.61 mg/dl and lactate clearance of 13.03%, whereas patients who died had a mean admission lactate value of 75.27 mg/dl and lactate clearance of 2.91%. The literature has reports both for and against serum lactate in predicting outcome in polytrauma patients. Several studies indicate that serum lactate at the time of admission and at serial intervals give a good prognostic value in major injuries, especially occult chest injury(36).

Blood lactate levels start rising as early as thirty minutes after major injury. Normal lactate levels depend on the age of the

individual and whether arterial or venous samples are used. Lavery et al found no difference between venous and arterial lactate levels(54). Significant lactic acidosis is said to have occurred when serum lactate value goes above 45 mg/dl or blood pH drops down to 7.35. Serial measurements of lactate were found to have a better prognostic value than a single measurement.

Manikis et al studied 129 patients admitted to ICU and found that there was a significant difference in the mean lactate levels in survivors as compared to non-survivors(55) . Abramson et al studied lactate clearance in 76 multi-trauma patients admitted to the ICU and concluded that the time needed to normalize lactate levels is a useful indicator in predicting prognosis in severely injured patients(37). Following major trauma, there is a surge of pro-inflammatory mediators, notably interleukin-6, until about the fourth day. The ‘second hit’ of any surgical intervention following the ‘first hit’ of the trauma itself is thought to have a deleterious effect on the outcome if any intervention is undertaken within this window. In a retrospective analysis of 4314 polytrauma patients, Pape et al. found that patients operated after 4^{th} day fared better than those operated between 2 to 4 days with respect to development of multi-organ failure(56).

In the setting of an acute lung injury, lactate levels increase as a result of endothelial damage, inhibition of pyruvate dehydrogenases and/or associated hypoxia and the resultant respiratory distress. Thus, elevated lactate values can be an important predictor of increased morbidity and mortality in trauma patients with an associated chest injury(57). In a study of 64 patients with torso trauma, Aslar et al. found lactate levels and the acute physiology and chronic health evaluation II (APACHE II) scores on admission to be predictive of survival(58).

Persistent lactic acidosis is associated with higher rates of multiple organ dysfunction syndrome and respiratory failure and death after major trauma(57). In our study, mortality rate for polytrauma patients with an elevated lactate was high.

In our series, patients who had significant rise of serum lactate (>45 mg/dl) at admission and remained elevated throughout had worse prognosis than whose lactate was significantly high at admission and normalized rapidly within 24-48 hours. Severity of injury and serum lactate levels were also more on patients with occult lung injury.

The main findings in our study are the following;

1) Initial blood lactate values and lactate clearances provide valuable information about the severity of the injury and predict outcome and mortality.

2) Initial lactate values & lactate clearances correlate well with vital parameters at the time of admission and with trauma scorings

3) Our study indicates that lactate clearance gives good prognostic value in polytrauma patients.

4) Lactate seems to be a good prognostic indicator in trauma even in patients with normal vital signs.

5) Lactate levels at admission correlate well with the quality of pre-hospital care and injury-admission interval.

In various types of shock, initial lactate levels rise due to increased production and also due to reduced excretion because of compromised renal function in polytrauma patients. In trauma patients, even the usage of alcohol or any other drug by the patient does not modify the predictive accuracy of initial blood lactate levels. In patients with hepatic contusion, initial lactate level may rise.

Several arguments say that initial lactate clearance was able to predict death(36). The initial blood lactate level correlates well with ISS. These two variables were also able to predict early death and the need for emergency procedures like external fixator application for Damage Control Orthopaedics. These were also able to predict number of days of ICU stay, which is important because, besides mortality itself, the duration of ICU stay might be a clinically and economically relevant criterion to assess morbidity of trauma patients. These were also found to predict massive haemorrhage assessed by the need of amount of blood transfusion.

We observed less amount of complications, like septicemia, ARDS, MODS, etc., less duration of ICU stay and faster clinical recovery in patients treated in form of damage control orthopaedics concept by applying external fixator and causing minimal secondary hit to the patients with occult hypoperfusion detected by high serum lactate and low lactate clearance.

In contrast, in patients with normal vital signs, initial blood lactate and lactate clearance did not add significant additional information to that provided by trauma scores like MGAP, RTS or TRISS. Thus, it proves that although lactate clearance may be useful to assess initial resuscitation, it cannot predict occult hypoperfusion

in normotensive individuals. Thus we accept the conclusion of other authors' opinion that renal artery or microcirculation blood flow should be measured to accurately detect occult hypoperfusion.

The evolution of mean blood lactate levels was significantly different in patients with normal versus abnormal blood lactate values at the time of admission(36). This means that the meaning of lactate clearance may not be the same in those patients having normal lactate value at admission.

We used logistic regression method to assess the additional value of blood lactate and lactate clearance to predict mortality. Using a reclassification method, comparison of models including initial serum lactate and lactate clearance versus ISS, RTS were found to be significant, implying that these two variables significantly increase the ability of trauma scorings to predict mortality(36). The odds ratio of initial lactate and lactate clearance are significant, using ISS as reference.

Some limitations of our study as follows:

First, this study was performed in adult population and thus may not apply to paediatric population. Second, our study was observational, could demonstrate association but not causality. Thus,

further studies are needed to demonstrate that interfering with lactate clearance using therapeutics actually modifies prognosis. The sample size is very less in our study and thus more cases over more period of time are needed to demonstrate the association of serum lactate values with the final outcome.

CONCLUSIONS

Serum lactate at the time of admission and lactate clearance at 6 hours can be a very good prognostic factor in predicting the morbidity and mortality in polytrauma and compound fractures. Serum lactate level at the time of admission and lactate clearance during the initial hours of admission can be a good predictor of patient's progress, development of complications, need for blood transfusion, need for continued monitoring and resuscitation in ICU and chances of death of the patient. As per our observation, serum lactate can also be useful in deciding about the timing of reconstructive surgery, i.e., implementing damage control orthopaedics by applying external fixator for pelvic and long bone fractures in patients with high serum lactate but with normal vital parameters resulted in good outcome. Persistent rise in lactate indicates ongoing shock or inadequate resuscitation and heralds the presence of even an occult chest injury.

Though some of our patients with elevated initial lactate values recovered well, probably due to better resuscitation, normal lactate values at the time of admission can be a useful means to identify low-risk trauma patients. Similarly, very high lactate at the

time of admission strongly predicted mortality in our study, although more sample size is needed to demonstrate the association.

Thus, a holistic approach with clinical, radiological and laboratory parameters coupled with reliable trauma scoring systems such as Injury Severity Score to guide the treatment aimed at early stabilization and mobilization of these kind of patients seem to be the emerging trend in the management of polytrauma patients. If facilities are available, we recommend more frequent estimation of lactate, calculate lactate clearance sequentially and give more attention to the patients who show persistently high lactate values even after 48 hours of admission. Lactate clearance provides additional predictive information to initial blood lactate levels and trauma scoring systems.

I suggest that, in future studies, it may be planned to use lactate values and lactate clearance in initial hours to assess the polytrauma patients to decide upon Early Total Care vs Damage control Orthopaedics and similarly in compound injury patients to decide upon early definitive fixation and temporary external fixation followed by late reconstructive surgeries.

CASE ILLUSTRATIONS

CASE-1 IP NO: 102010

- 29 year old male with H/O fall from height
- Injury – Admission Interval: 41 hours
- Pre-hospital care: IV Fluids, Antibiotics & Analgesics
- Diagnosis: Right sacral fracture with pubic diastasis – vertically unstable type of pelvic injury; ISS – 25
- Vital parameters:

	Sys BP mmHg	**MAP mmHg**	**SpO2 %**	**HR /mt**	**RR /mt**	**Temp °f**	**GCS**	**Urine output/hr**
At admission	130	96	94	86	16	98	15	50
After 6 hours	130	96	95	84	14	99	15	55
After 24 hours	130	96	98	80	14	99	15	55

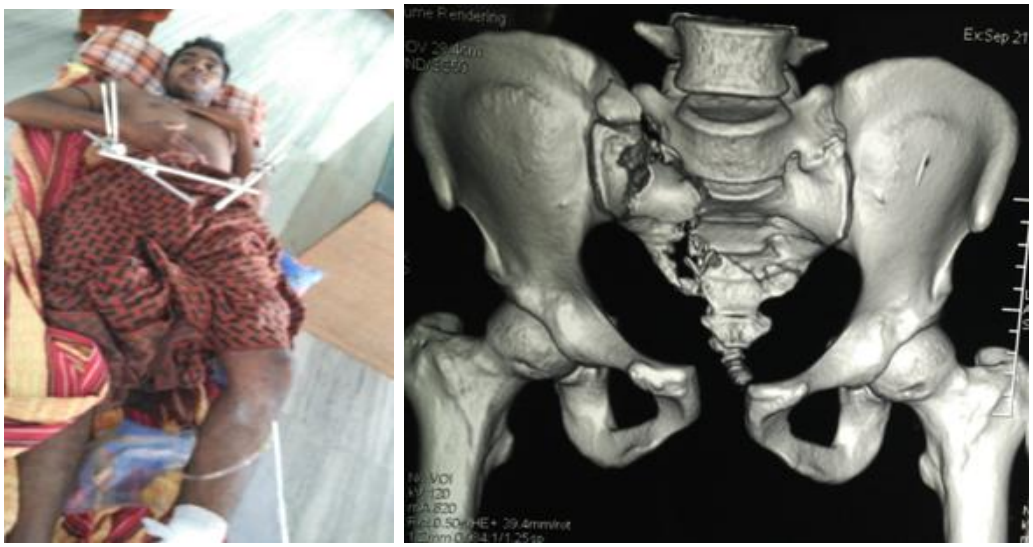
- Blood parameters:

	pH	**Hbg %**	**Glu mg %**	**Ur mg %**	**Cr mg %**	**Na+ mEq/L**	**K+ mEq/L**	**Lactate mg%**	**% Lactate Clearance**
At admission		10.8	77	16	0.8	134	3.4	24	18.8
After 6 hr								20	41.4
After 24 hr		11.6	80	23	1	135	4	18	27.9

- Treatment given: Pelvic External Fixator on day 1, Blood transfusion, Internal fixation on day 20, Antibiotics

- Comment: Occult hypoperfusion is identified early with high serum lactate; patient responded well with proper resuscitation and external fixator & recovered without any complications.

Clinical Picture & Pre op CT



X ray with External Fixator

CASE-2 IP NO: 97865

- 54 year old female with H/O RTA
- Injury – Admission Interval: 6 hours
- Pre-hospital care: IV fluids (1.5 L); Oral (0.5 L)
- Diagnosis: B/l Shaft of femur fracture, Both bones leg Fracture Rt side, Laceration lt leg
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	94	74	90	112	22	98	15	40
After 6 hours	100	80	93	104	17	99.2	15	45
After 24 hours	110	90	95	96	15	100	15	50

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission		10.1	90	27	0.7	145	4	32.4	22.2
After 6 hr								25.2	19.8
After 24 hr		11.5	92	41	1	143	3.9	20.2	37.7

- Treatment given: B/l Skeletal traction, Wound debridement, Antibiotics, Analgesics, Lt Femur Plating on day 26
- Complications: Superficial Wound infection, AKI, LRI, prolonged hypotension & ICU stay (4 days)
- Comment: Patient might be benefitted with early femoral external fixator as per DCO concept.

Clinical Picture & X-ray Rt Leg

X ray Rt Femur & x ray Lt Femur

CASE-3 IP NO: 103285

- 20 year old male with H/O TTA
- Injury-Admission interval: 6 hrs
- Pre-hospital care: IV fluids-2 L
- Diagnosis: Crush injury both legs with B/l Comp gr3C BB # Leg
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	84	64	88	120	28	97.6	15	30
After 6 hours	90	70	92	102	20	98.2	15	40
After 24 hours	96	76	94	94	18	99	15	45

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission		7.1	124	32	1	125	5	42	17
After 6 hr								35	23.4
After 24 hr		7.9	125	49	1.4			23	33.5

- Treatment given: B/l Guillotine Through Knee Amputation in 6 hrs of admission, Antibiotics, Analgesics, Tetanus immunoglobulin
- ICU stay-36hrs
- Complications- AKI, Superficial Wound infection
- Comment: Grossly elevated lactate at admission warranted continued resuscitation even after attaining haemodynamic stability; AKI resolved with intensive care

Wound Picture of Both Legs

CP after amputation & X-rays of both legs showing BB fractures

CASE-4 IP NO: 102074

- 79 year old male with H/O RTA
- Injury – Admission Interval: 14 Hours
- Pre-hospital care: IV fluids, wound wash, Antibiotics
- Diagnosis: Comp Gr2 Tibial Plateau Fracture Rt Leg
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	98	78	92	96	20	98	15	40
After 6 hours	108	88	94	88	18	98.4	15	45
After 24 hours	114	94	95	84	16	98.2	15	55

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission		11.8	156	26	1	132	4.2	28	10
After 6 hr								23	47
After 24 hr		12	142	30	1.1			18	28.9

- Treatment given: Knee Spanning External Fixator in 8 hrs of admission, Antibiotics, Analgesics, LCP fixation on day 22.
- Complications: Nil
- Comment: Early temporary stabilization washed out lactate thereby addressing occult hypoperfusion quickly which prevented complications

Clinical Picture ***Pre op x ray***

Post op x ray

CASE-5 IP NO: 86475

- 32 year old alcoholic male with H/O RTA, neglected for 24 hrs
- Injury-Admission Interval: 24 hrs
- Pre-hospital care: IV fluids (2 L)
- Diagnosis: Gas Gangrene of Rt upper limb with extensive myonecrosis; ISS-75; MESS-12;

Vital parameters	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	80	60	82	132	32	102	14	25
After 6 hours	70	50	82	124	30	105	13	20
After 24 hours								

Blood parameters	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission	7.3	7.5	74	44	2	120	5	85	-3.76
After 6 hr								88	
After 24 hr									

- Treatment given: Rt Forequarter Amputation in 3 hrs of admission, Antibiotics, Intubation, Ventilator support
- Complication: Death in 6 hrs
- Comment: Very high lactate and negative lactate clearance correlates with rapidly progressing septicemia, hypoperfusion, MODS.

CASE-6 IP NO: 81368

- 50 year old male with H/O RTA
- Injury- Admission Interval: 8 hrs
- Pre-hospital care: IV fluids (2 L), NPO
- Diagnosis: Rt Frontotemporal haematoma, Shaft of Femur#, BB Leg, Bimalleolar #
- Vital parameters:

	Sys BP	Mean AP	SpO2	HR	RR	Temp	GCS	Urine output/hr
At admission	100	80	95	94	24	98	14	40
After 6 hours	108	86	97	84	20	99	14	50
After 24 hours	112	94	98	82	18	100	15	60

- Blood parameters:

	pH	Hb	Glu	Ur	Cr	Na+	K+	Lactate	Lactate Clearance
At admission	7.39	5.8	119	109	3	137	5	29	17.2
After 6 hr	7.39							24	34.5
After 24 hr	7.4	7	72	107	2.8			19	20.8

- Treatment given: External Fixator application on day 1, Serial wound debridements, Antibiotic coated K nailing for Femur & LCP for proximal tibia on day 46, Appropriate antibiotics
- Complications: ICU stay- 48hrs, Deep wound infection, AKI on CKD.
- Final outcome: Late recovery (60 days of hospital stay)
- Comment: Early external fixation as per damage control concept limited blood loss enabling faster haemodynamic recovery; serum lactate values correlate.

Clinical Picture & Pre op x ray- Tibia

Post op x ray – Tibia & X ray Rt Femur

CASE-7 IP NO: 99124

- 48 year old unknown male with H/O TTA
- Injury-Admission Interval: 6 hours
- Pre-hospital care: IV Fluids (3.5 L)
- Diagnosis: Mangled left thigh with femur #, ISS-25
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	84	64	85	124	30	96	15	20
After 6 hours	88	68	89	114	28	97.2	ET	25
After 24 hours								

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission	7.4	5.4	74	44	2	124	5	74	1.6
After 6 hr	7.3	5.6						75	
After 24 hr									

- Treatment given: Lt Hip Guillotine disarticulation after 2 hours of admission, Antibiotics, Analgesics, Tetanus Immunoglobulin, Intubation and ventilator support

- Complication: Death in 8 hours

- Comment: Very high lactate at admission and negative lactate clearance correlates with outcome.

Clinical Picture

CASE-8 IP NO: 84503

- 22 year old male with H/o RTA
- Injury – Admission Interval: 2 hrs
- Pre-hospital care: IV fluids (2 L), Oral fluids (1 L)
- Diagnosis: Comp GrIIIA Supracondylar Femur Fracture Lt, Calcaneal Fracture Rt,
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	96	78	88	98	28	98	15	30
After 6 hours	104	84	92	96	25	99	15	40
After 24 hours	112	86	95	84	22	99	15	50

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission		8.4	154	29	1	134	4	26	6.92
After 6 hr								24	26.15
After 24 hr		8.8	124	32	1.4			19	20.66

- Treatment given: Wound debridement, External Fixator for Femur, K Wire for Calcaneum on day 1, Antibiotics, Analgesics, LCP for Femur on day 32
- Complications: Superficial Wound infection
- Final outcome: Late recovery (45 days of hospital stay)
- Comment: Proper resuscitation and early external fixation corrected lactate level within 24 hrs and prevented major complications.

Clinical Picture & Pre op CT

X ray # Supracondylar Femur Lt, Calcaneum # Rt Pre & Post op

CASE-9 IP NO: 96642

- 26 year old male with H/O RTA
- Injury- Admission Interval: 3 hrs
- Pre-hospital care: IV fluids (1 L), Oral fluids (0.5 L)
- Diagnosis: Schatzker type VI Tibial Plateau # Lt Side
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	100	80	92	89	24	98	15	40
After 6 hours	110	90	94	86	20	98.8	15	50
After 24 hours	114	94	95	82	16	99	15	55

- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission		9.2	114	26	1	129	4	25	15.9
After 6 hr								21	27
After 24 hr		9.4	124	28	1.2			18	13.2

- Treatment done: External Fixator on day 1, Antibiotics, analgesics, LCP fixation on day 19

- Final outcome: Early recovery (30 days of hospital stay)

- Comment: External fixator was applied to prevent further damage to skin and soft tissues and to stabilize the fracture which in turn quickly washed out lactate from blood. Patient recovered without any complications.

Clinical Picture & Pre op Xray of Tibial Plateau #

Post op Xray of Tibial Plateau # Lt

CASE-10 IP NO: 86404

- 65 year old female with H/O RTA
- Injury- Admission interval: 8 hrs
- Pre-hospital care: IV Fluids (3 L), Oral(1 L)
- Diagnosis: Comp GrIIIB BB # Both Leg, Comp Humerus # lt
- Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission	96	78	88	100	24	98	14	35
After 6 hours	104	88	92	96	20	99	15	45
After 24 hours	112	86	95	88	16	100	15	50

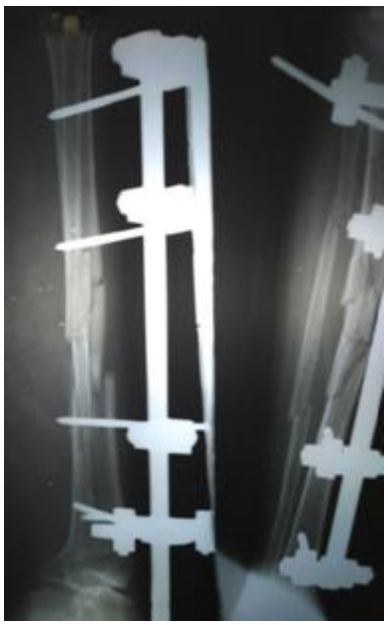
- Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na+ mEq/L	K+ mEq/L	Lactate mg%	% Lactate Clearance
At admission	7.4	8.4	145	42	1.3	135	4.5	34	6.47
After 6 hr	7.4							32	7.55
After 24 hr	7.4	9.4	168	38	1.3			29	13.53

- Treatment given: External fixator for left leg on day 1, serial wound debridements, Antibiotics, Analgesics
- Complications: Septicemia, AKI, 72 hours of ICU stay
- Final outcome: Late recovery (58 days of hospital stay)
- Comment: Although the initial lactate levels were not very much high, the poor lactate clearance in this patient explains about the ongoing septicemia. Anticipating and addressing earlier would have prevented the complications.

Clinical Picture & Xray Lt Arm showing Humerus Fracture


Pre op X ray showing # BB B/l Leg & Post op X ray Lt leg

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ANNEXURES

MANGLED EXTREMITY SEVERITY SCORE

Score for Compound fractures to decide on Amputation/Salvage.

≥ 7 - Primary Amputation

< 7 - Limb Salvageable

THE SKELETAL/SOFT TISSUE INJURY

Low energy injury (stab wound, closed fracture, low velocity, (Gunshot wound)	1
Medium energy injury (Open fracture, multiple fractures, dislocation)	2
High energy injury (shotgun wound, high velocity gunshot wound, crush injury)	3
Very high energy injury (as above, but with gross contamination, soft tissue avulsion)	4

LIMB ISCHEMIA

Pulse reduced or absent, normal perfusion less than 6 hours	1
More than 6 hours	2
Pulseless, paraesthesiae, reduced capillary refill less than 6 hours	2
More than 6 hours	4
cool, paralyzed, insensate limb less than 6 hours	3
more than 6 hours	6

SHOCK

BP more than 90 mm Hg	0
Transient hypotension	1
Persistent hypotension	2

AGE

< 30 years	0
30 – 50 years	1
> 50 years	2

ABBREVIATED INJURY SCALE

An anatomical scoring system to measure the severity of injury of each anatomical region.

1 - Minor

2 - Moderate

3 - Serious

4 - Severe

5 - Critical

6 - Unsurvivable

INJURY SEVERITY SCORE (ISS) & NEW INJURY SEVERITY SCORE (NISS)

The Injury Severity Score (ISS) is an anatomical scoring system for patients with multiple injuries.

AIS allocated for each body region.

ISS = Sum of squares of top 3 AIS.

An example of the ISS calculation is shown below:

Region	Injury description	AIS	Square	Top three
Head & Neck	Cerebral contusion	3	9	
Face	No Injury	0	0	
Chest	Flail chest	4	16	16
Abdomen	Minor contusion of Liver complex Rupture spleen	5	25	25
Extremity	Fractured femur	3	9	9
External	No injury	0	0	

Injury severity score - 50

NISS = Sum of squares of top 3 AIS regardless of body region.

S No	Name	A/S	IP No	Inj-Adm Int	MOI	Diagnosis	ISS	MESS	Vitals & Blood parameters																										
									SBP			MAP			SpO2			HR			RR			Temp			GCS			Urine Output	Blood pH			Hemo
									0 hr	6 hr	24hr	0 hr	6 hr	24 hr	0 hr	6 hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	1-6 hr	0hr	6hr	24hr	0hr
1	Subramani	70/M	81821	9hrs	RTA	segmental Rt Femur & Lt BB Leg Fracture, Rt frontoparietotemporal SDH	25	-	90	100	110	70	80	90	90	95	95	102	96	82	24	20	18	98.4	98.8	99	9	11	11	120	-	-	-	8.2
2	Udayakumar	24/m	81788	8 hrs	RTA	Pneumomediastinum & Pneumothorax Lt, knee dislocation lt	17	-	90	100	110	70	80	90	85	90	95	104	98	84	34	24	20	98.4	98.6	99.4	15	15	15	150	-	-	-	8.4
3	Sekar	50/m	81368	8 hrs	RTA	Rt frontotemporal hematoma, shaft of femur, BB leg, bimalleolar & comp 1st MTB fracture	20	-	100	108	112	80	86	90	95	97	98	94	84	82	24	20	18	98	99	100	14	14	15	150	7.39	7.39	7.4	5.8
4	Ayyappan	40/m	82822	4 hrs	RTA	comp monteggia fracture dislocation Rt	9		130	130	130	104	104	104	98	98	98	92	84	80	18	16	14	98	99	99	15	15	15	180	-	-	-	9.4
5	Pounkumar	20/m	82619	3 hrs	RTA	Multiple brain contusions, blunt injury abdomen, fracture left humerus, rt femur, b/l pubic rami	48	-	90	96	102	70	76	80	85	90	92	96	88	84	34	28	25	97.4	100	103	5	5	5	100	7.35	7.35	7.32	8.2
6	Karthick	36/m	83613	4 hrs	RTA	comp gr3b BB lt leg, facial lacerations	18	3	120	120	120	100	100	100	96	98	98	84	80	80	28	20	18	98	99	100	15	15	15	180	-	-	-	9.4
7	Ganapathy	23/m	83644	8 hrs	RTA	Comp gr3b BB # rt leg	9	2	124	124	120	98	96	92	96	95	95	92	90	86	22	20	18	98	99	100	15	15	15	180	-	-	-	8.4
8	Harigopalakrishnan	30/m	84547	3 hrs	TTA	Crush injury lt leg with BB #	16	8	94	100	110	74	80	84	90	94	95	102	94	90	28	25	23	98	99	99	15	15	15	180	-	-	-	8.2
9	Sathish	22/m	84503	2 hrs	RTA	Comp gr3a Supracondylar # lt femur, Comp gr3a calcaneal # Rt, 3rd MTB # Rt	16	2	96	104	112	78	84	86	88	92	95	98	96	84	28	25	22	98	99	99	15	15	15	150	-	-	-	8.4
10	Sendhil	35/m	84527	5 hrs	RTA	Comp gr2 BB lt leg, shaft of femur segmental # lt	16	3	100	108	116	82	88	90	92	95	95	104	96	84	28	22	18	98	99	100	15	15	15	150	-	-	-	8.2
11	Balaji	30/m	84343	4 hrs	RTA	Compgr1 Fracture shaft of femur rt	9	2	102	110	120	82	92	94	92	95	95	102	94	82	28	22	18	98	99	100	15	15	15	150	-	-	-	10.2
12	Sathishkumar	24/m	86420	5 hrs	RTA	Crush injury rt arm & lt leg,	16	8	90	100	108	70	80	88	90	92	95	104	96	82	28	22	17	98	99	100	15	15	15	150	-	-	-	6.4
13	Karuppiah	30/m	86032	6 hrs	RTA	Comp gr3a supracondylar # rt femur, Bimalleolar # rt, lt temporal bone #	25	3	90	100	108	70	80	88	89	94	96	104	96	84	28	24	22	98	99	100	12	13	14	130	-	-	-	9.8
14	Manivannan	39/m	86013	12 hrs	Fall	Comp gr3a calcaneal # rt foot	9	4	110	116	120	84	90	92	95	98	98	92	84	84	16	14	14	98	99	100	15	15	15	150	-	-	-	9.2
15	Ponnusamy	52/m	85654	6 hrs	RTA	Comp gr3a supracondylar # Rt femur	16	4	100	106	112	80	90	92	90	95	95	99	92	84	24	18	16	98	99	100	15	15	15	130	-	-	-	9
16	Veerasamy	65/m	85321	4 hrs	RTA	Closed Supracondylar # Rt Femur with Schatzker type VI Tibial Plateau #, Crush injury rt foot	16	4	110	116	124	84	96	98	92	95	96	98	94	84	26	22	18	98	99	100	15	15	15	130	-	-	-	8.4
17	Thangavel	49/m	85985	12 hrs	RTA	Comp gr3b BB # Lt leg with schatzker type VI Tibial Plateau	16	4	96	100	110	76	80	90	90	96	95	102	95	84	28	20	18	98	99	100	15	15	15	120	-	-	-	8.2
18	Hariraman	24/m	86475	24 hrs	RTA	Comp Gr3c Humerus # with gas gangrene	75	12	80	70		60	50		82	82		132	124		32	30		102	105		14	13		100	7.25			7.5
19	murugesan	35/m	87012	4 hrs	RTA	Comp gr3a Supracondylar # Rt femur	16	2	102	108	114	82	88	94	90	94	95	102	96	94	24	20	16	98	99	100	15	15	15	140	-	-	-	8.8
20	Ayyappan	44/m	87121	5 hrs	RTA	Comp gr3a Monteggia # dislocation lt, Comp gr2 BB # Rt leg	16	4	108	112	120	88	92	94	92	95	95	96	94	84	20	18	16	98	99	100	15	15	15	150	-	-	-	8.4
21	Ayyappan	19/m	87132	3 hrs	RTA	Comp Gr3a Monteggia # equivalent with segmental Ulna # Lt	16	4	100	108	120	80	88	94	92	95	95	98	94	90	22	18	18	98	99	100	15	15	15	150	-	-	-	8.9
22	Velayudham	33/m	87452	4 hrs	RTA	Comp gr3a BB # rt leg	9	3	110	114	120	80	94	96	95	96	96	92	86	82	18	16	16	98	99	100	15	15	15	180	-	-	-	9.2
23	devendran	35/m	87654	3 hrs	RTA	Comp gr3a BB # Lt leg	9	3	112	120	124	92	94	98	95	98	98	94	84	78	18	15	15	98	99	100	15	15	15	180	-	-	-	9.4
24	singaram	65/m	87702	2 hrs	RTA	Closed Intertrochanteric, BB Rt Leg, Distal radius Lt #	9	4	106	112	114	86	92	94	90	92	92	94	82	78	16	14	14	98	99	100	15	15	15	180	-	-	-	8.2
25	Ganesan	39/m	87745	3 hrs	RTA	Comp gr3b BB # Lt leg lower 3rd	16	3	110	118	124	80	98	98	92	94	94	98	84	82	18	14	14	98	99	100	15	15	15	180	-	-	-	8.4
26	Mahendran	28/m	87789	8 hrs	RTA	Comp gr3b BB # Rt Leg, Shaft of femur # Rt, Patella #	20	4	90	100	108	70	80	88	88	90	94	102	94	90	24	20	18	98	99	100	15	15	15	200	-	-	-	8.1
27	Devaki	65/f	86404	8 hrs	RTA	Comp gr3b # B/L BB # Leg, Comp Humerus # Lt	41	5	96	104	112	76	88	86	88	92	95	100	96	88	24	20	16	98	99	100	14	15	15	180	7.38	7.4	7.42	8.4
28	Ayyappan	45/m	86870	2 hrs	RTA	Comp gr3b Shaft of humerus # Lt with radial nerve palsy, Lt Zygomaxillary #	24	4	98	106	114	78	88	88	89	92	96	103	98	89	25	20	14	98	99	100	13	14	15	150				9.4
29	David	23/m	87783	3hrs	RTA	Comp gr2 Tibial Plateau# with compartment syndrome	16	4	104	112	120	86	92	94	92	95	96	94	86	76	18	15	14	98	99.4	100.2	15	15	15	200				10.2
30	Sakthivel	25/m	89913	3hrs	RTA	supra condylar # lt femur with distal BB leg #, Lt frontal sinus #, lt clavicle #	20	3	106	114	118	86	86	100	91	94	96	92	84	78	16	14	14	98	99.2	99.6	15	15	15	150				9.8
31	Ganesan	38/m	89471	7hrs	RTA	Comp gr3b Segmental BB # Rt leg	9	3	108	112	124	88	86	98	92	95	97	94	88	84	15	14	14	98	98	99	15	15	15	200				9.4
32	Thangasamy	80/m	87795	6hrs	RTA	Comp gr3b BB # lt leg, dentatoalveolar #	18	4	104	110	118	84	90	98	88	90	92	98	94	88	19	17	15	98	99.2	99.8	15	15	15	150				9.4
33	Krishnan	35/m	89921	7 hrs	RTA	Crush injury rt foot, metatarsal fractures, # lt bimalleolar	9	3	108	116	126	88	92	98	90	94	95	92	86	82	15	14	14	98	98.8	99.4	15	15	15	200				9.8
34	Sumanraj	21/m	89758	4hrs	RTA	Crush injury both foot with matatarsal fractures	9	3	106	114	120	86	94	94	92	96	97	98	92	86	16	14	14	98	98.9	99.3	15	15	15	200				9.4
35	Natarajan	60/m	89089	12hrs	RTA	Comp gr3b BB # Lt leg, Supracondylar # Rt femur	18	4	98	104	112	78	88	92	90	94	96	98	92	84	18	14	13	98	98.8	99.3	15	15	15	200				9.4
36	Raja	26/m	90313	10hrs	RTA	Comp gr2 Monteggia # dislocation Rt, inferior pole of patella# RT	16	3	100	106	114	80	86	86	92	94	95	98	94	88	18	15	14	98	98	99.3	15	15	15	200				9.8
37	Sengalan	38/m	90645	4hrs	RTA	Shaft of Femur# Rt, Comp gr3b distal BB # Rt leg	18	4	96	102	114	76	82	94	90	94	96	102	96	88	18	15	14	98	98.8	99	15	15	15	180				8.8
38	Ramu	26/m	91602	8hrs	RTA	Crush injury both foot with matatarsal fractures, Lt rib fracture with hemothorax	18	3	98	106	118	78	84	96	89	93	94	104	96	86	22	18	16	98	99.6	100.2	15	15	15	150				10.2
39	Natarajan	60/m	89089	4hrs	RTA	Comp gr3b BB # lt leg & comp gr3b supracondylar # rt femur	25	7	94	102	112	74	82	94	92	95	96	102	95	88	18	14	14	98	99.8	101.2	15	15	15	200				9.4

S No	Name	A/S	IP No	Inj-Adm Int	MOI	Diagnosis	ISS	MESS	Vitals & Blood parameters																										
									SBP			MAP			SpO2			HR			RR			Temp			GCS			Urine Output	Blood pH			Hemo
									0 hr	6 hr	24hr	0 hr	6 hr	24 hr	0 hr	6 hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	0hr	6hr	24hr	1-6 hr	0hr	6hr	24hr	0hr
40	Sampath	56/m	91007	14 hr	RTA	Type3 APC Pelvic & Lt Acetabulum #, Compgr3b BB # Lt leg, BB forearm #, Nasal bone #, B/l Hemothorax	34	4	86	92	100	66	72	80	85	89	94	124	102	94	24	19	17	97.6	99.4	100.5	14	15	15	120	7.4	7.38	7.37	9.6
41	Iqbal	35/m	90981	2 hrs	RTA	Compgr3b BB # Rt Leg, Mandible #	18	3	100	104	110	80	84	80	91	95	96	98	88	78	20	18	16	98	98.8	99.4	15	15	15	150				9.8
42	Rajamani	75/f	92408	8 hrs	RTA	Comp gr3b BB # Rt Leg	9	3	104	112	118	84	86	92	92	96	96	98	90	84	18	16	14	98	98.8	99.6	15	15	15	150				9.2
43	Kumar	61/m	92107	4 hrs	RTA	Lt Clavicle, Neck of Humerus, Ulna #, Multiple ribs # with Hemopneumothorax	18		96	102	110	76	82	84	89	93	95	102	96	90	18	15	14	98	99	100.2	15	15	15	120	7.38	7.42	7.4	9.2
44	Amitbasha	58/m	92731	4 hrs	RTA	Comp gr3b Supracondylar # Rt Femur	16	3	100	108	116	80	88	96	90	94	96	104	98	92	17	14	14	98	99	100.2	15	15	15	150				9.8
45	Karthick	22/m	92567	4 hrs	RTA	Comp gr3c BB # Lt leg	16	8	98	104	110	80	86	84	92	95	96	102	95	88	18	16	14	98	98.8	99.4	15	15	15	160				9.7
46	Rangarajan	44/m	92513	6 hrs	Fall	L1,L2 #, Rt SPR, IPR, Sacral #, Calcaneal, Talus #	18		102	108	116	82	90	96	90	94	96	94	88	82	18	16	15	99.2	99.2	99.8	15	15	15	200				10.2
47	Ramasamy	37/m	92542	7 hrs	RTA	Comp gr3b Segmental shaft of Femur # Rt, Pubic diastasis, SI jt disruption Rt	32	4	92	100	108	72	80	88	90	94	95	104	98	94	20	16	14	98	98.8	99.6	15	15	15	200				10.4
48	Yuvraj	38/m	95181	4 hrs	RTA	Comp Gr3c Supracondylar # Lt femur, Comp gr2 Shaft of femur # Rt, Comp Olecranon # Rt, Distal radius # Lt	41	9	88	96	104	68	76	84	85	89	92	112	102	96	25	20	15	98	99.8	101	15	15	15	120	7.4	7.32	7.31	8.4
49	W Chan	27/m	95471	8 hrs	RTA	Comp gr3b humerus # Lt, # Dislocation Rt Hip, Supracondylar femur #, BB leg # Rt	32	4	94	102	114	74	82	94	88	92	94	102	92	88	24	20	18	98	99.4	99.8	15	15	15	150				9.2
50	Vasanthan	52/m	95377	4 hrs	RTA	Comp Gr1 Tibial Plateau# Rt, Central # Hip dislocation Lt	18	3	98	106	118	80	88	94	87	93	97	104	96	88	20	18	16	98	99.4	100.6	15	15	15	150				10.4
51	sakthivel	25/m	85593	6 hrs	RTA	Lt Clavicle, Supracodylar femur & BB leg #, lt frontal sinus #	18	3	100	108	118	80	86	100	87	92	93	104	98	90	18	16	14	98	99.4	100.4	15	15	15	150				9.4
52	gopi	28/m	89898	4 hrs	TTA	# Clavicle Rt, Comp gr2 # BB Rt Leg, Rt Hemothorax	25	3	96	104	112	76	86	84	88	92	94	102	96	87	18	16	15	98	99.2	100.4	15	15	15	180				9.8
53	ravi	55/m	95155	6 hrs	RTA	Comp gr3b Shaft of femur # Lt	16	3	100	108	116	80	88	94	90	92	94	96	94	88	18	16	14	98	99.4	100.8	15	15	15	150				9.6
54	janakiraman	52/m	95404	6 hrs	RTA	Comp gr3b BB # Rt Leg, Comp gr1 Supracondylar # Rt Femur	25	4	96	104	112	76	84	92	90	94	96	98	94	88	18	16	14	98	99.4	100.8	15	15	15	150				9.8
55	mahendrasingh	45/m	95009	4 hrs	RTA	Comp gr3b BB # Lt leg with calcaneum#	16	3	102	112	120	82	94	100	90	92	94	96	92	86	18	14	14	98	98.8	99.4	15	15	15	180				10.2
56	egambaram	49/m	95560	6 hrs	RTA	Comp gr1 lat epicondyle & Radial head #, Lt hemopneumothorax	18	3	94	102	114	74	84	94	92	94	95	102	96	88	18	14	14	98	99.8	100.8	15	15	15	200				10.2
57	bhuvaneshwari	56/f	97869	6 hrs	RTA	Comp gr3b BB # lt leg	16	3	98	104	108	80	82	88	85	89	92	103	96	92	17	14	14	98	99.4	100.2	15	15	15	120	7.38	7.3	7.3	8.4
58	anandhan	45/m	96633	8 hrs	RTA	BB # Rt Leg, Blunt injury abdomen, Hemoperitoneum,Bladder injury	34		94	100	108	76	80	88	85	89	92	112	100	94	20	18	16	98	99.8	101.2	15	ET	ET	150	7.37	7.24	7.14	9.2
59	jana	16/m	97838	6 hrs	RTA	Pubic diastasis with SI jt disruption, Blunt injury abdomen, Ileal injury	41		92	102	108	74	80	88	84	92	94	120	108	98	24	18	18	97.8	100	102	15	ET	ET	150	7.39	7.31	7.29	7.4
60	murugesan	58/m	93685	8 hrs	TTA	B/l Crush injury leg with BB leg #, peritoneal injury	50	10	86	94	100	66	76	80	86	91	95	114	102	96	23	19	16	98	100.2	101.4	15	15	15	120				8.4
61	ganesan	27/m	97748	6 hrs	RTA	# Shaft of femur with Comp gr3c BB # rt leg, Bladder injury	41	9	88	96	104	68	74	82	87	92	95	104	98	88	21	19	16	98	99.2	101.2	15	15	15	150	7.4	7.38	7.37	8.4
62	anjali	54/f	97865	6 hrs	RTA	B/l Shaft of femur #, BB leg # rt	25		94	100	110	74	80	90	90	93	95	112	104	96	22	17	15	98	99.2	100.2	15	15	15	180				10.1
63	Vijay	29/m	102010	41hrs	Fall	Rt Sacral fracture with Pubic diastasis	25		130	130	130	96	96	96	94	95	98	86	84	80	16	14	14	98.4	99	99	15	15	15	150				10.8
64	Lingeshwaran	20/m	98015	6hrs	TTA	B/l Crush injury leg compGr3C BB # Leg	32	10	84	90	96	64	70	76	88	92	94	120	102	94	28	20	18	97.4	98.2	99	15	15	15	100				7.1
65	Kandasamy	79/m	102074	14hrs	RTA	CompGr1 Tibial Plateau Fracture Rt Leg	16	3	98	108	114	78	88	94	92	94	95	96	88	84	20	18	16	98.4	98.4	98.2	15	15	15	180				11.8
66	unknown	48/m	99124	6hrs	TTA	Mangled left thigh with Femur#	25	12	84	88		64	68		85	89		124	114		30	28		96.4	97.2		15	ET		80	7.35	7.3		5.4
67	Manojkumar	26/m	96642	4hrs	RTA	Schatzker type VI Tibial Plateau # Lt side	9		100	110	114	80	90	94	92	94	95	89	86	82	24	20	16	98.4	98.8	99	15	15	15	200				9.2
68	Udayakumar	34/m	78245	6hrs	RTA	B/L Comp Gr3B BB # Leg with posterior dislocation of left hip	41	5	86	90	96	66	72	76	88	90	92	104	99	89	28	25	22	97.2	98	99.5	14	14	ET	100	7.35	7.32	7.3	7.2
69	Kumar	54/m	99879	6hrs	RTA	Lt Hemopneumathorax with Comp gr3b BB # Lt leg	25	4	88	98	104	68	78	84	88	90	92	114	108	98	24	20	16	97.4	98.2	99	15	15	15	120				8.2

S No	Hb%		Bl glucose		Sr urea		Sr Creatinin		Na	K	Sr Lactate mg/dl			Lactate clearance %			Procedures done		Blood transfused	Lt of Ventilation	Lt of ICU stay	Complications	Pt outcome
		24hr	0hr	24hr	0hr	24hr	0hr	24hr			0hr	6hr	24hr	0-6	0-24	6-24 hrs	within 24 hrs	after 24 hrs					
1		8.4	160	140	24	25	1.2	1.1	130	4	35	31	24	11.50	31.50	22.50	-	IL nailing for femur & tibia	2	-	24 hrs	delayed recovery from head injury	Recovered with sequlae
2		8.5	120	114	24	24	1.1	1.1	132	4.1	27	23	19	11.50	29.63	17.39	ICD, Ext fixator		2	-	36 hrs	ARDS	Late recovery
3		7	119	72	109	107	2.8	2.8	137	5.3	29	24	19	17.24	34.48	20.83	External fixator	Femur nailing, Tibia plating	3	24 hrs	24 hrs	wound infection, AKI	Late recovery
4		9.8	110	104	23	22	1.1	1.1	132	4.1	18	16	15	11.11	16.67	6.25	External fixator & K wiring		1	-	-	-	Early recovery
5		8.4	102	140	24	35	1.1	1.9	130	4	70.2	65.4	65	6.84	7.41	0.61	Intubation,ICD, fracture splintage		4	72 hrs	3 days	Sepsis, MODS	Recovered with sequelae
6		9.6	110	108	23	22	1	1.1	135	4.2	17.3	17.5	18	-1.16	-4.05	-2.86	External fixator, facial suturing		1	-	12 hrs	wound infection	Early recovery
7		9.1	94	110	33	34	1.2	1.4	131	4.1	35.2	28	19.2	20.45	45.45	31.43	external fixator		1	-	12 hrs	wound infection, AKI	Late recovery
8		9	102	94	24	30	1	1.2	129	4.2	24	22.4	19.4	6.67	19.17	13.39	BK Amputation		3	-	18 hrs	wound infection, AKI	Early recovery
9		8.8	154	124	29	32	1.2	1.4	134	4.2	26	24.2	19.2	6.92	26.15	20.66	External fixator, k wire fixation	Femur LCP	2	-	12 hrs	Wound infection, AKI	Early recovery
10		8.6	124	102	32	34	1.2	1.3	129	4.4	36	29.4	24.4	18.33	32.22	17.01	Splintage	IL nailing for femur & tibia	2	-	48 hrs	Prolonged hypotension	Late recovery
11		9.4	135	114	33	30	1.1	1.3	134	4.1	24	21	18.6	12.50	22.50	11.43	-	IL nailing for femur	1	-	24 hrs	-	Early recovery
12		8.2	124	114	28	40	1.1	1.5	132	4.2	35	32	24	8.57	31.43	25.00	BE Amputation, wound debridement		4	-	24 hrs	AKI	Late recovery
13		10.2	156	147	29	34	1.1	1.5	134	3.9	26	24	18	7.69	30.77	25.00	External fixator, k wire fixation		1	-	24 hrs	Wound infection	Early recovery
14		9.4	124	132	24	25	1.1	1.2	135	4	22	19	18.4	13.64	16.36	3.16	Wound debridement, External fixator		1	-	12 hrs	Wound infection	Early recovery
15		8.6	170	168	33	37	1.2	1.4	127	4.4	28	24.4	20.2	12.86	27.86	17.21	Wound debridement, External fixator	LCP for Femur	2		24 hrs	Wound infection, AKI	Late recovery
16		8.2	164	154	40	44	1.4	1.7	130	4.5	34.5	30.2	26.4	12.46	23.48	12.58	Wound debridement, K wire fixation	ORIF with LCP for femur & Tibia, SSG	2	-	36 hrs	Wound infection, AKI, LRI	Recovered with sequelae
17		8	110	145	38	33	1.1	1.2	130	4.1	24	22.2	19.2	7.50	20.00	13.51	Wound debridement, External fixator		2	-	24 hrs	Wound infection	Early recovery
18			74		44		1.5		120	5.4	85.2	88.4		-3.76	100.00	100.00	Shoulder disarticulation		4	7 hrs	-	Septicemia, Gas gangrene	Death within 2 days
19		9	140	138	25	24	1.1	1.2	132	4.1	20.2	18.4	16	8.91	20.79	13.04	Skeletal traction	IL Nailing	1	-	12 hrs	-	Early recovery
20		8.8	142	134	24	22	1.1	1.1	135	4.2	34	29.2	24.4	14.12	28.24	16.44	Wound debridement, Closed reduction of elbow	IL Nailing for Tibia, Plating for ulna & wiring for radial head	2	-	36 hrs	Wound infection, Knee stiffness	Late recovery
21		8.5	132	134	25	24	1.2	1.1	135	4.1	24.4	22.8	20.2	6.56	17.21	11.40	Wound debridementelbow spanning external fixator & k wires					Elbow stiffness, infection	Recovered with sequelae
22		9.4	135	132	24	25	1.1	1.1	136	4.2	21.2	20.4	18.8	3.77	11.32	7.84	Wound debridement, external fixator		1		12 hrs	Wound infection	Early recovery
23		9.8	134	142	25	24	1.1	1.2	134	4.1	20.8	19.4	18.4	6.73	11.54	5.15	Wound debridement, external fixator	IL nailing for tibia	1		6 hrs	-	Early recovery
24		8.4	158	170	38	42	1.5	1.8	126	4.5	25.2	21.4	20.2	15.08	19.84	5.61	POP	DHS, Tibial nailing	1		6 hrs	CKD	Late recovery
25		8.6	145	158	27	28	1.1	1.2	129	4.2	24	21.2	19.2	11.67	20.00	9.43	Wound debridement, external fixator		2		12 hrs	Wound infection	Early recovery
26		9.2	152	148	32	38	1.1	1.4	135	4.2	25	20.2	15.5	19.20	38.00	23.27	Wound debridement, external fixator,	femur nailing	2		48 hrs	Superficial infection	Early recovery
27		9.4	145	168	42	38	1.3	1.3	135	4.5	34	31.8	29.4	6.47	13.53	7.55	Wound debridement, External fixator		3		72 hrs	Septicemia, AKI	Late recovery
28		9.2	123	107	24	28	1	1.1	134	4.1	33.4	31.2	27.4	6.59	17.96	12.18	Wound debridement, External fixator, Radial nerve repair		2		48hrs	Septicemia	Recovered with sequelae
29		9.4	114	105	25	28	1	1.2	135	4.2	27	21	15.1	22.22	44.07	28.10	Fasciotomy, Wound debridement, knee spanning external fixator		2		48hrs	Wound infection, Knee stiffness	Recovered with sequelae
30		9.6	71	75	24	25	1	1.1	134	4.1	28	25.4	22	9.29	21.43	13.39	Skeletal traction	ORIF with LCP	2		24hrs	Wound infection	Early recovery
31		9.6	84	98	24	24	0.9	1.1	135	4.1	21.2	19.2	18.8	9.43	11.32	2.08	Wound debridement, External fixator		1		24 hrs	Wound infection	Early recovery
32		9.8	145	157	34	39	1.1	1.4	135	4.2	41.2	35.4	29.2	14.08	29.13	17.51	Wound debridement, External fixator		2		24hrs	Wound infection, AKI	Late recovery
33		9.6	142	134	24	26	1	1.3	135	4.1	24.5	22.3	20.2	8.98	17.55	9.42	Wound debridement, K Wire fixation		1		28hrs	Wound infection	Early recovery
34		9.8	138	142	26	29	1	1.2	136	4.2	25.2	21.4	19.3	15.08	23.41	9.81	Wound debridement, K Wire fixation		1		18hrs	Wound infection	Early recovery
35		9.8	135	145	24	29	1.1	1.3	135	4.1	37	32.4	27	12.43	27.03	16.67	Wound debridement, b/l knee spanning external fixator	Femur LCP	2		48hrs	Wound infection, Knee stiffness	Recovered with sequelae
36		9.4	108	124	26	29	1	1.2	138	4.3	34	26.4	22.4	22.35	34.12	15.15	Wound debridement, External fixator, Tube slab		3		36hrs	Wound infection, RSD	Late recovery
37		9.4	125	104	24	29	1	1.1	134	4.1	34	25.2	19.4	25.88	42.94	23.02	Wound debridement, External fixator	Femur nailing	2		48 hrs	Wound infection	Early recovery
38		10.4	104	124	24	25	1	1.1	135	4.2	28	24	22.4	14.29	20.00	6.67	Wound debridement, K Wire fixation, ICD insertion		2		48hrs	Wound infection, ARDS	Late recovery
39		9.2	81	95	24	32	1	1.3	135	4.3	39	32.2	27.4	17.44	29.74	14.91	Wound debridement, external fixator	Ak Amputation Lt	3		72 hrs	Septicemia, Crush syndrome	Late recovery

S No	globin		Bl glucose		Sr urea		Sr Creatinin		Na	K	Sr Lactate mg/dl			Lactate clearance %			Procedures done		Blood transfused	Lt of Ventilation	Lt of ICU stay	Complications	Pt outcome
																	within 24 hrs	after 24 hrs					
	24hr	0hr	24hr	0hr	24hr	0hr	24hr			0hr	6hr	24hr	0-6	0-24	6-24 hrs								
40	9.2	140	102	25	33	1	1.2	134	4.4	40.2	36.5	32.4	9.20	19.40	11.23		Wound debridement, Pelvic & Ankle spanning external fixator		4	24hrs	96hrs	Septicemia,	Recovered with sequelae
41	9.6	134	124	25	29	1.1	1.3	135	4.2	30	27.4	22.4	8.67	25.33	18.25		Wound debridement, external fixator		2		24 hrs	Wound infection	Early recovery
42	8.8	187	198	30	36	1.2	1.4	128	4.6	34	28.4	24.4	16.47	28.24	14.08		Wound debridement, Ankle spanning external fixator	Flap cover	2		48 hrs	Wound infection, AKI	Late recovery
43	9.4	124	132	24	30	1.1	1.2	129	4.2	32	27.5	23.4	14.06	26.88	14.91		ICD,	Plating for ulna & Humerus	2		72 hrs	Prolonged ICU care	Late recovery
44	9.6	129	132	25	30	1.2	1.3	135	4.1	24	22.3	19.2	7.08	20.00	13.90		Wound debridement, Knee spanning external fixator	SSG	2		48 hrs	Wound infection	Early recovery
45	9.5	132	124	24	28	1	1.2	136	4.2	26.4	23.4	21.4	11.36	18.94	8.55		BK Amputation	Revision Amputation	2		48hrs	Wound infection	Recovered with sequelae
46	10.8	175	187	24	28	1	1.2	135	4.2	24	22.2	19.4	7.50	19.17	12.61		POP, Pelvic binder	Posterior Stabilization	2		24 hrs		Early recovery
47	9.8	140	132	26	29	1	1.2	136	4.2	38.4	34.2	32.6	10.94	15.10	4.68		Wound debridement, Pelvic external fixator, Knee & Ankle spanning fixator	Femur plating	3		72 hrs	Wound infection, prolonged hypotension	Late recovery
48	8.8	145	134	27	42	1.1	1.6	128	4.5	44.1	42.1	38	4.54	13.83	9.74		AK Amputation Lt, Olecranon K Wire	Femur nailing rt	3	12 hrs	96 hrs	Wound infection, Crush syndrome, AKI	Recovered with sequelae
49	9.4	123	104	24	28	1	1.2	135	4.2	43.2	36.4	30.2	15.74	30.09	17.03		Wound debridement, Humerus external fixator	Percutaneous Acetabulum screw fixation, LCP for femur, Tibial Nailing	2		48 hrs	Wound infection, Prolonged ICU stay	Late recovery
50	9.6	104	84	23	29	1	1.1	141	3.9	32.2	27.4	24.2	14.91	24.84	11.68		Pin traction	Tibial LCP, Acetabulum fixation	1		36 hrs	Prolonged ICU care	Late recovery
51	9.8	140	135	24	29	1.1	1.2	142	3.8	32.4	24.4	19.4	24.69	40.12	20.49		Pin traction	ORIF	1		24 hrs		Early recovery
52	10.2	104	99	24	28	1	1.1	135	4	33.2	24.2	19.4	27.11	41.57	19.83		ICD, POP	Tibial Nailing	2		36 hrs	Wound infection, Prolonged ICU stay	Early recovery
53	9.8	85	96	24	30	1	1.3	138	4.2	11.6	10.5	9	9.48	22.41	14.29		Wound debridement,External fixator		1		24 hrs	Wound infecton	Early recovery
54	10.2	157	164	27	32	1.1	1.3	129	4.1	43.1	35.4	29.2	17.87	32.25	17.51		Wound debridement, External fixator		2		48 hrs	Wound infection, Prolonged ICU stay	Late recovery
55	10.4	104	120	24	28	1	1.2	135	4.2	32.1	24.1	19.2	24.92	40.19	20.33		Wound debridement, External fixator	Skin cover	1		24 hrs	Wound infection	Early recovery
56	10.4	114	124	24	29	1.1	1.3	135	4.3	34.2	29.4	23.4	14.04	31.58	20.41		ICD, Wound debridement, K wire & External fixation		2		24 hrs	Wound infection, RSD	Late recovery
57	8.6	158	166	28	39	1.2	1.5	129	4.4	72.4	67.5	64.2	6.77	11.33	4.89		Wound debridement, External fixator		2		48 hrs	Septicemia, crush syndrome	Death within 2 days
58	8.4	145	124	24	34	1.1	1.5	128	5	68.2	64.3	58.2	5.72	14.66	9.49		Laparotomy, Bladder repair		5	10 days	10 days	Septicemia, MODS,	Death after 2 days
59	8.2	124	134	24	38	1.1	1.5	124	4.8	68.2	42.4	33.6	37.83	50.73	20.75		Pelvic external fixator, Laparotomy, Ileal resection & Anastomosis		4	7 days	10 days	Prolonged ventilatory care	Recovered with sequelae
60	8.2	134	126	24	36	1.1	1.4	129	4.1	69.2	62.4	57.4	9.83	17.05	8.01		Wound debridement, B/l Guillotine BK amputation, Peritoneal repair	Revision Amputation	4	5 days	7 days	Wound infection, Prolonged stay	Recovered with sequelae
61	8.9	104	96	20	38	0.9	1.3	132	4.1	42.3	33.2	24.4	21.51	42.32	26.51		Bk amputation, bladder repair	Revision through knee, Femur plating	4		5 days	Wound infection, prolonged stay	Recovered with sequelae
62	11.5	90	92	27	41	0.7	1	145	4.1	35.2	27.5	23.4	21.88	33.52	14.91		Traction	ORIF	3		3 days	Wound infection	Late recovery
63	11.6	77	80	16	23	0.8	1	134	3.4	24	20	18	18.78	27.89	41.43		Pelvic External fixator	ORIF	2		24hrs	Nil	Early Recovery
64	7.9	124	125	32	39	1.2	1.3	125	4.5	42.4	35.2	23.4	16.98	33.52	23.40		B/L Guillotine Through knee amputation	Revision	4		36hrs	AKI, Wound infection	Late recovery
65	12	156	142	26	30	1	1.1	132	4.2	28.2	23.4	18.2	9.95	28.87	47.12		Knee Spanning External Fixator	ORIF	2		18hrs	Nil	Early recovery
66	5.6	74		44		1.6		124	5.4	74.2	75.4		1.61				Hip Guillotine Disarticulation		6	8hrs	8hrs	Crush syndrome, MODS,	Death within 2 days
67	9.4	114	124	26	28	1.2	1.2	129	4.1	25.2	21.2	18.4	15.87	13.21	27.00		Knee Spanning External Fixator	ORIF	1		6hrs	Nil	Early recovery
68	7.4	114	104	24	38	1.2	1.4	128	4.1	65.4	62.2	54.4	4.90	12.54	1.68		Wound debridement, External fixator, Hip reduction		3	10 days	14 days	Septicemia, ARDS, MODS, Prolonged Intubation	Recovered with sequelae
69	8.4	104	108	26	28	1.1	1.3	129	4.1	34.2	29.4	22.4	14.04	34.50	23.81		Wound debridement, External Fixator, ICD	IL Nailing	3		48hrs	Wound infection	Early recovery

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PROFORMA

Name/Age/Sex/IPNo. :

Date/Time of admission/Injury-Admission Interval:

Pre-hospital care:

Diagnosis:

Trauma scoring (ISS/MESS):

Vital parameters:

	Sys BP mmHg	MAP mmHg	SpO_2 %	HR /mt	RR /mt	Temp °f	GCS	Urine output/hr
At admission								
After 6 hours								
After 24 hours								

Blood parameters:

	pH	Hb g%	Glu mg%	Ur mg%	Cr mg%	Na^+ mEq/L	K^+ mEq/L	Lactate mg%	% Lactate Clearance
At admission									
After 6 hr									
After 24 hr									

Procedure done/Date :

Length of Ventilation/ICU/Hospital stay:

Any complications:

Final clinical outcome:

Date of discharge/death:

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராயச்சியின் தலைப்பு

கடுமையான மற்றும் பல்வேறு காயமுற்ற நோயாளிகளின் இறுதி நிலையை, வளர்ச்சிதை மாற்ற காரணிகள் மூலம் முன்கணிக்கும் ஆய்வு.

ஆய்வு நிலையம் : முடநீக்கியல் மற்றும் விபத்தியல் துறை,
சென்னை மருத்துவக் கல்லூரி சென்னை – 3.

பங்கு பெறுவரின் பெயர் :

பங்குபெறுபவரின் எண் :

பங்குபெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கின்றேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் 'இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிகிறேன். ☐

இந்த தொழில்நுட்ப முறை ஒற்றுக்கொள்ளப்பட்ட ஒன்று என்பதையும் இதனால் உடலுக்கு எந்தவிதமான உபாதைகளும் இருக்காது என்பதை அறிந்துகொண்டு இந்த ஆய்வில் பங்குபெற முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம்..... தேதி.....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம்..... தேதி.....

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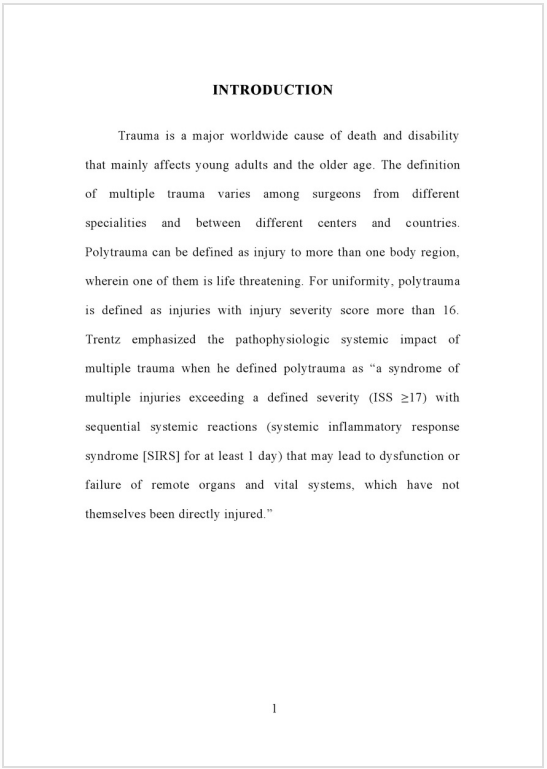

INTRODUCTION

Trauma is a major worldwide cause of death and disability that mainly affects young adults and the older age. The definition of multiple trauma varies among surgeons from different specialities and between different centers and countries. Polytrauma can be defined as injury to more than one body region, wherein one of them is life threatening. For uniformity, polytrauma is defined as injuries with injury severity score more than 16. Trentz emphasized the pathophysiologic systemic impact of multiple trauma when he defined polytrauma as "a syndrome of multiple injuries exceeding a defined severity (ISS ≥17) with sequential systemic reactions (systemic inflammatory response syndrome [SIRS] for at least 1 day) that may lead to dysfunction or failure of remote organs and vital systems, which have not themselves been directly injured."

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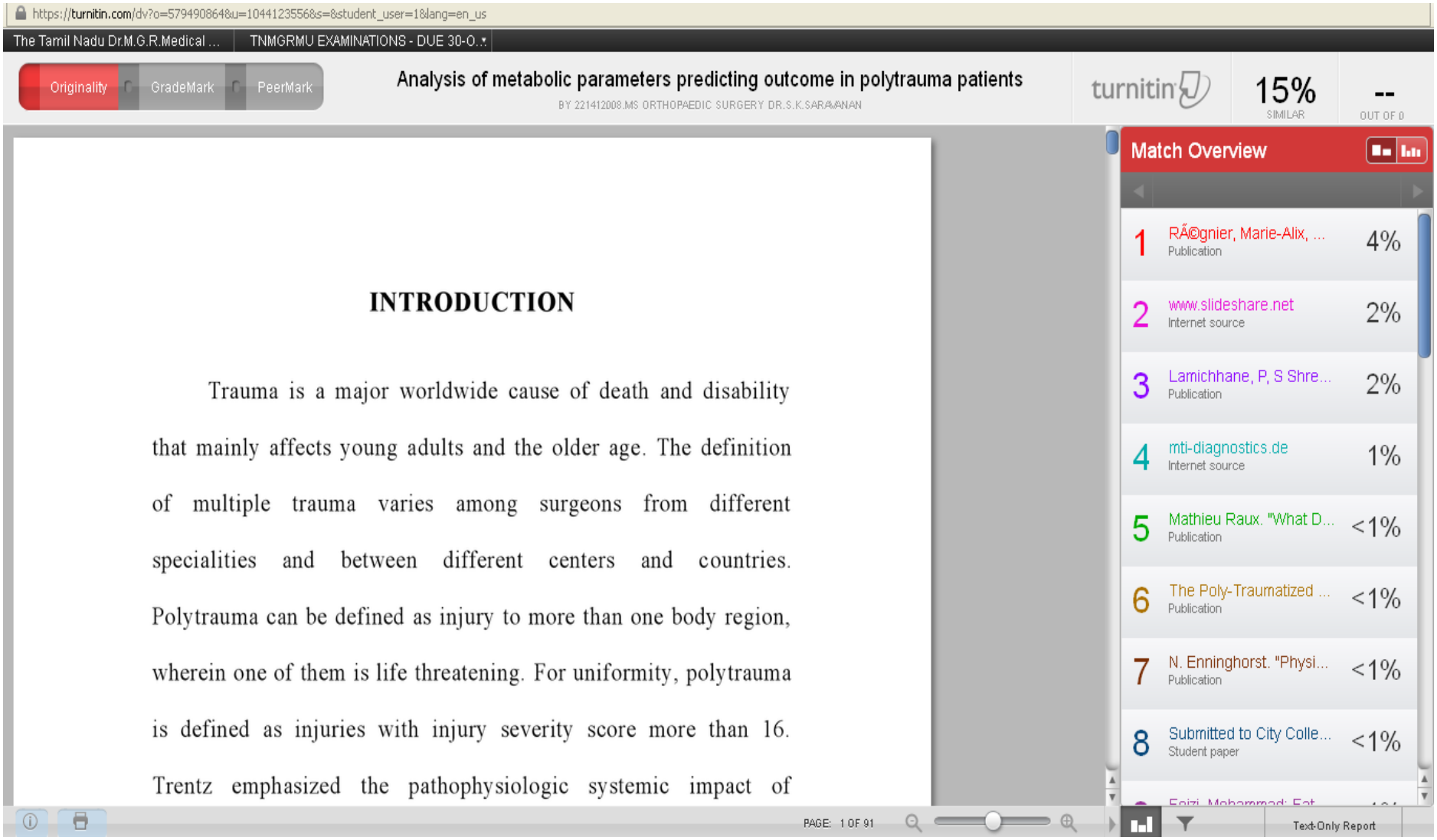
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CERTIFICATE OF APPROVAL

To
Dr.S.K.Saravanan
Postgraduate M.S.(Orthopaedics)
Madras Medical College
Chennai 600 003

Dear Dr.S.K.Saravanan,

The Institutional Ethics Committee has considered your request and approved your study titled **"Metabolic parameters predicting outcome in polytrauma patients" No.28072015.**

The following members of Ethics Committee were present in the meeting held on 07.07.2015 conducted at Madras Medical College, Chennai-3.

1. Prof.C.Rajendran, M.D.,	: Chairperson
2. Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4. Prof.B.Vasanthi, M.D., Professor Pharmacology, MMC	: Member
5. Prof.P.Ragumani, M.S., Professor, Inst.of Surgery, MMC	: Member
6. Prof.Md.Ali, M.D., D.M., Prof. & HOD of Medl.G.E., MMC	: Member
7. Prof.Baby Vasumathi, Director, Inst.of O&G, Ch-8	: Member
8. Prof.K.Ramadevi, Director, Inst.of Biochemistry, MMC	: Member
9. Prof.Saraswathy, M.D., Director, Inst. Of Pathology, MMC	: Member
10. Prof.Srinivasagalu, Director, Inst.of Inter Med. MMC	: Member
11. Thiru S.Rameshkumar, B.Com., MBA	: Lay Person
12. Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
13. Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003